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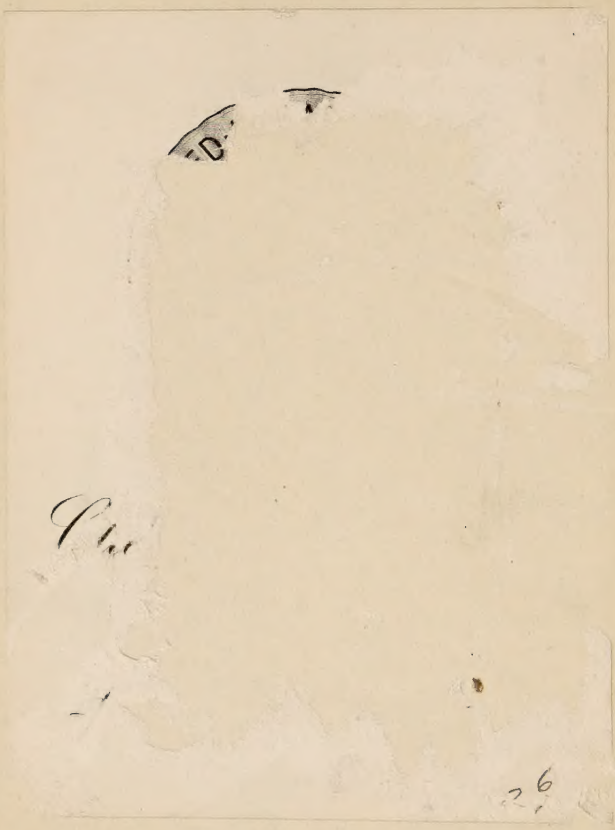

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TEN POST-GRADUATE LECTURES

TEN

POST-GRADUATE LECTURES

delivered before the Fellowship of Medicine

AT THE HOUSE OF

THE ROYAL SOCIETY OF MEDICINE

1919—1920

WITH A PREFACE BY

THE RIGHT HON. SIR CLIFFORD ALLBUTT P.C., K.C.B., M.D., F.R.S.

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1922

COURSE OF PHYSICS
OF
PHILADELPHIA

CONTENTS.

PAGE

PREFACE. By the Right Hon. Sir CLIFFORD ALLBUTT, P.C., K.C.B., M.D., F.R.S. ... ix

SYPHILIS AND INSANITY. By Sir GEORGE H. SAVAGE, M.D., F.R.C.P. 1

THE PROGNOSIS OF EXOPHTHALMIC GOITRE, By Sir WILLIAM HALE-WHITE, K.B.E., M.D., F.R.C.P. 19

GRAVE FAMILIAL JAUNDICE OF THE NEWLY BORN. By Sir HUMPHRY ROLLESTON, K.C.B., M.D., F.R.C.P. 31

THE VALUE OF COMBINED TREATMENT, WITH SPECIAL REFERENCE TO SURGERY, ELECTRICITY AND X-RAYS. By FRANCIS HERNAMAN-JOHNSON, M.D. 40

ON THE AFTER-EFFECTS AND SO-CALLED AFTER-EFFECTS OF ANÆSTHETICS. By J. D. MORTIMER, M.B., F.R.C.S. 59

CHRONIC PAROXYSMAL TRIGEMINAL NEURALGIA AND ITS TREATMENT. By WILFRED HARRIS, M.D.Cantab., F.R.C.P. 68

MORBID MENTAL GROWTHS. By Sir GEORGE H. SAVAGE, M.D., F.R.C.P. 83

PYORRHŒA ALVEOLARIS AND ITS RELATIONSHIP TO DISEASE. By N. MUTCH, M.A., M.D., F.R.C.P. 101

DISABILITIES OF THE FEET DUE TO STATIC OR MECHANICAL CAUSES. By W. H. TRETHOWAN, M.B., B.S., F.R.C.S. ... 116

DEFORMITIES AND DISABILITIES OF THE FEET DUE TO PARALYSIS. By W. H. TRETHOWAN, M.B., B.S., F.R.C.S. 149

NOTE.

While it is true that "good wine needs no bush," it is desirable and useful for the best of wines to be labelled with their vintages by a competent connoisseur, and therefore, while I knew that the quality of the Fellowship post-graduate vintage now offered to a wider public was of fine quality, I made bold to ask the connoisseur (*facile princeps*) of the medical profession, Sir Clifford Allbutt, to label the bins. With that kindly helpfulness which is only equalled by his ability, he at once consented to do so, and I am very proud to be able to present the result as an introduction to our first volume, for which we are indebted to the enterprise of Messrs. Bale.

J. Y. W. MacAlister.

PREFACE.

THE fault of the utilitarian is not his bent to utility, but his narrow vision fixed upon immediate utilities; he fails to see that even to accomplish immediate ends he must often fetch a wider compass. Yet however disinterested our search may be, it is determined, nevertheless, by its ends, whether we perceive them or not. A general movement, of which we may hardly be aware, leads us along certain ways. The English doctor has been too individual; he has gathered experience, even richly, but it has stayed with him, and been limited by his limits; the harvests have vanished with the death of the reapers. But the hope of a continually progressive civilization, a progress without periods of darkness, suffering and reaction, depends upon the comparatively modern methods of *records,* the recording of experiences not only of individuals but also of companies of workers, whereby the harvests of observation are gathered in, and the work of many husbandmen multiplied and blended. In its largest sense then utility means a more comprehensive vision; by the flow of the larger streams the nearer and narrower channels will be filled up.

It is by such tendencies, some conscious some unconscious, that the natural sciences have grown so rapidly during the last few centuries. For as men found themselves congregating into larger and larger

societies, they found also that only by scientific knowledge, and by pooling their several resources in conversation and record, were such civic aggregations possible. Even earlier, as men began to move abroad and to traverse wilderness and sea, astronomy became necessary; and so forth in the case of the more and more complex sciences, they arose, too often sadly belated, as each became indispensable for the supply of its own peculiar aids.

Unfortunately for city life a science much more intricate and difficult than astronomy or mechanics was indispensable; namely, biology; for it is only by the applications of biology that in the aggregations of men those periods of eclipse, misery and devastation which are due to biological disorders can be avoided. We hope now that no longer shall cities be wiped out, as great centres of civil life have been; for instance by malaria in South Italy. Yellow fever and perhaps plague should soon follow malaria into disregard if not into oblivion. Against such diseases as these, which have depopulated cities, the individual physician, however sagacious, has been helpless.

It is then by companies of men, such as the Fellowship of Medicine, Research Committees, Medical Associations, Hospital Research Units, Colleges, and so forth—each covering its peculiar ground, and usually recording its own work—only by such common action is city and suburban life to be made possible. Nay, however well brigaded, physicians cannot operate efficiently over large densely populated or widely scattered areas without laboratories, central and provincial; nor indeed with-

out some measures of official control, whether we call it governmental or co-operative. Unfortunately such organization brings with it the evils of all bureaucracies, the loss of elasticity and the stereotyping of methods. It is of vital importance therefore, while joining in the call to united action, to cherish also the more spontaneous and various agencies which, like the Fellowship of Medicine, the sundry Medical Societies, The British Medical Association, and so forth, spring directly out of our own life and business, and are self-controlled. Thus a reasonable independence may be conserved without waste of effort.

The perusal of the lectures in this volume will go far to convince the most self-contained of practitioners that in our time without periodical class teaching and co-operative practice he cannot efficiently equip himself, nor do full justice to his patients. Again, only by wide collection and comparison of records can the Health Officer deal effectively with the health of a community. In every Lecture of this volume we begin to see that in all grave disorders, in many indeed of a transient kind, something more than the service of the single-handed family physician or surgeon is required. From them also we learn more and more clearly that to the end of our lives we are students, and indeed hungry half-filled students. After all we come short of intellectual food. From these lectures, and the more of them which will appear, we shall learn to perceive more precisely just where and in what phase of a disorder the aid of colleagues, and of costly apparatus, is required.

The Lectures of Sir George Savage remind us of

what we have lost in the death of one of the happiest, wittiest, kindliest and, in his own subject, most experienced members of our profession. To those many graduate hearers, whose opportunities of studying mental disease are comparatively infrequent, these lessons must have been invaluable. I would especially emphasize for family practice his warning on the disconcerting difficulty in many cases of early diagnosis between Neurasthenia and General Paralysis of the Insane. Before the day of Wassermann I remember a most anxious case of this difficulty in a very important personage. The uncertainty had been laid before the patient's friends, and soon afterwards it was resolved by the secret departure of the patient into foreign lands, and the prompt arrival of a wire for a large sum of money. Sir George seems disposed to think that in early stages of syphilitic infection, and in ataxic patients, some indefinite signs of mental disorder may often be observed; an observation which supports the remarks that I made at the British Medical Association at Newcastle on the early access of the parasite or its toxins to the brain. And in congenital cases we shall agree with him that the Hutchinsonian phenomena are often absent. Also that children born of a parent before the appearance of General Paralysis may be normal, but not those born afterwards. Again, he rightly reproves us for calling Jacksonian convulsions "Epilepsy," which is the name of another series of a different nature. In discussing the effect of a shock of any kind in arousing General Paralysis one would have liked to argue against him

that the shock acted rather by weakening resistance—local or general—than by awakening the malignancy of the parasite. And again, that under Delusional Insanity, rather than date its origin from a distressing dream, one should regard the dream as the sign of a balance failing first during the relaxation of sleep.

Sir William Hale-White endows practitioners, whose work lies in smaller fields, with the richer materials of a large hospital for the furtherance of prognosis, which can be founded only on large numbers. Yet it was the doctrine of the Hippocratic school that in Prognosis lay the strength and the warrants of Medicine. After reading Sir William's lecture on the prognosis of Graves' Disease we feel fortified at least, if not certified, in a working opinion on the probabilities of success by medical and by surgical means respectively. The result comes out better for medical management than many of us might have expected. We learn furthermore that the Prevention of Disease is not peculiarly a municipal or State system, but depends on principles which are as valid for the individual as for the community, and differ only in the means and methods of application.

Sir Humphry Rolleston's Lecture, on the grave form of jaundice in the newborn (mortality 77 per cent.), brings together the remarkable tale of twenty-five family groups. He adopts the hypothesis that this disease is due to some toxæmia in the mother which sets up a cholangitic cirrhosis in her child; and thus opens out a broad view of pathology.

A like broad view is opened in therapeutics by the brightly-written Lecture of Dr. Hernaman-Johnson on the combination of electrical, surgical and X-ray treatment in certain disabilities. For instance, in the treatment of scrofulous glands in the neck, for which the operation of Teale and Allbutt had become almost universal,[1] the Lecturer now advises first a previous reduction of the swellings by X-rays; surgery being left for the removal of the remnant. But time after time we had to urge that in the imperfectly successful cases our rules were often imperfectly followed: e.g., to operate early before the skin is implicated; and secondly, which is far from easy, that no tunnels leading to deeper glands —often very small or concealed under the sterno-mastoid or other structure—should be left unexplored. Again, Dr. Hernaman-Johnson's words on the treatment of the effects of Anterior Poliomyelitis are hopeful. When I entered upon clinical studies among out-patients these cases of palsy were put aside on what was called the "Incurable bench."

Dr. Mortimer's Lecture on the after-effects of anæsthesia is timely; he rightly urges a preciser discernment of causes—e.g., of vomiting, too often taken to be unavoidable. But among the causes of vomiting ought not commencing sepsis to be reckoned?

Dr. Harris's striking summary of his well-known methods in Trigeminal Neuralgia must have called

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[1] Mr. Rushton Parker had performed this operation once six years before, but he did not follow it up.

forth the admiration of his hearers. It has been the privilege of few men to have brought full relief to sufferings so intolerable. How successful his method has been I have had occasion personally to witness in two or three cases. The Lecturer alluded to patients under the age of 30 (e.g., aged 21 and 22), and made some interesting suggestions as to possible obscure peripheral sepsis. In one bilateral case he operated on both sides with relief, and with quite a fair motor result. He thinks that the alcohol does not destroy all the motor fibres in the ganglion. His test by novocain of a good shot into the ganglion is happy. I agree with Dr. Harris that the only drug of any efficacy in this malady is gelsemium. In one case in an elderly man I achieved a complete cure by it alone—to my great astonishment. In another case, a woman, great relief was obtained, but I lost sight of the patient.

It was interesting to hear what further news Dr. Mutch, in his Lecture on Pyorrhœa Alveolaris, had to give us concerning the application of scientific methods to the parrot cry of "intestinal toxins." His conception of a balance of bacterial reactions in the normal bowel is notable, and in its way may be of like significance to the balance of hormones. A practical warning, therefore, not to stir up the microbes too suddenly or blindly by purgatives, massage, or exercises is useful. At the same time we are glad to be informed that, as an intestinal antiseptic colloidal sulphur and a combination of ammonium ichthyo-sulphurate with proteins has been found efficacious.

I have left myself little room to touch upon Mr. Trethowan's two able and elaborate articles on disabilities of the feet. It would be hard to think of a subject more helpful to the general practitioner, who thus learns to discern the various kinds of such lameness, and how to seek the means of cure. His surgery I dare not venture even to commend; but if it is as good as his neurology it will do; for it seems to be founded no less accurately on anatomical facts. Both Mr. Johnson and Mr. Trethowan speak not only of restoration of muscular powers but also of restoration of growth of limbs; for, as he says, the arrest of growth bears no direct proportion to the palsy.

Such lectures as these, reinforced by the personal influence of the teachers, must be a godsend to graduates who wish to keep abreast of the medicine and surgery of the day; especially if illuminated, as no doubt they were, by clinical demonstrations. The Fellowship of Medicine is to be congratulated on so eminent a part of its still more extensive labours.

CLIFFORD ALLBUTT.

SYPHILIS AND INSANITY.

By Sir GEORGE H. SAVAGE, M.D., F.R.C.P.[1]

Consulting Physician for, and Lecturer on Mental Diseases, Guy's Hospital.

In selecting a subject for a post-graduate lecture I have felt that the great essential is to give as nearly as possible an account of the knowledge on the subject up to date, but beside this that it should represent the work or the experience of the lecturer himself. As to the first, there can be no doubt about the great interest and importance of the subject; as to the latter, I have been writing on the subject since 1888, and I am therefore old enough to have seen the whole of the ideas of syphilis changed. Wilks, in the early sixties, used to say that up to then the profession seemed to think that syphilis was only manifest in the skin and the terminals of the alimentary tract; now we know that no part of the body is free from danger, and that the nervous system is especially liable to the most serious harm.

In treating the subject to-day, first I have placed before you what I might call my present creed. This does not cover all the ground nor do I intend to go through the table in any degree of completeness, but I recognize that it is well to have the country charted so that you may recognize where you are.

[1] Deceased since the delivery of this lecture.

I shall start with considering syphilis as a psychical and secondly as a physical cause of mental disorder.

I have for years felt the difficulty in using the word "cause," for I hardly ever recognize one antecedent as the only real cause of any disorder. This is true of syphilis. Take for instance, as a fact that it is a certain associated cause of general paralysis of the insane, yet there are native races who are said to be saturated with syphilis, yet free from general paralysis of the insane.

First, as to the moral effect of syphilis in producing insanity. I have met with a fair number of persons who, without even having committed any immoral act, have become obsessed by the feeling that they had syphilis. Such cases have generally suffered from some sexual disorder. I have met with the ascetic celibate and the youthful masturbator with such ideas, and what has interested me much has been that with decay as with evolution of the sexual desires, similar disorder of feeling and similar delusions as to contagion have arisen. In these cases in the earlier or slighter stages I have only found out by careful observation that the patients were in the habit of destroying as much as they could of paper or clothing which came in contact with their skins. Such cases too may be given to constant washing of their hands or wearing gloves by day and night. Such fear of contagion may occur with other diseases as well as syphilis and I have met with delusion as to hydrophobia.

The more common form of moral disorder is well named syphilophobia, and follows either the actual infection or at least having run the risk of contagion.

Take an example: A man now over 60 when about 30 contracted syphilis in quite a mild form, but he has never got over the dread of the disease, and every illness or malaise which he has he attributes to syphilis. I have known him consult specialists of many kinds for his various troubles—the dermatologist for acne on his back and neurologist for vague pains in his head. In his case temporary relief seems to follow each fresh consultation, but the future is almost certain to be one of confirmed *hypochondriasis*. Yet in this case there is little danger to society.

A similar case was that of a German who, having suffered from syphilis, went for complete treatment to Aix. He returned to England fairly contented, but with some slight ailment. He was convinced that his old enemy was pursuing him, and once more he went to Aix. Later with the development of Wassermann diagnosis each illness of any kind was followed by a visit to the Continent, so that his blood and spinal fluid should be tested. Even a negative reaction failed to satisfy him, and he would once more go over to Aix. His future too is that of the hypochondriac who, occupied with his own introspection, does not suffer so much as his friends. The above have been slight cases, the next was a more serious one.

A man of middle age who had held a good Civil

Service appointment, contracted syphilis, and was fully treated by the greatest authority of his time. For a time he seemed satisfied, but slowly dwelling on slight feeling of sexual weakness, he became convinced that he was constitutionally a wreck, and that the old syphilis was the cause. He got leave of absence for treatment, went to the Continent, but with little faith in the result, and in the end he killed himself.

In all the above instances the personal feeling was the dominant one, but the most dangerous cases are those who think that others recognize that they are suffering from the disease, and shun them. I shall have to refer to similar cases in which the disfigurement due to scars gave rise to morbid self-consciousness; at present I will give an example of the groups of cases. A military man who had served with distinction, had in early life contracted syphilis. He had been treated, and reported as cured, but whenever he got run down the fear of the disease returned. When I first saw him, he was in abject dread. He would not go into his club because he felt other men either avoided him or looked suspiciously at him, and he felt they were whispering and suggesting that he should be removed from the club. He was to a certain extent relieved by having a trained attendant, but even then he would only go out after dark, and to non-frequented places. One day he rushed away from his companion, and going to his flat, which was on the third floor, he waited in fear, feeling certain that he was being followed and that a crowd was

pursuing him to lynch him. He told me after he heard them coming and threw himself from the window, and though he lived for a few hours, he died. I have met with several similar instances, and they all were intensely suicidal.

A somewhat difficult class to explain is that of some women with a limited knowledge of what venereal disease is, who have developed the delusion that they have the disease and may have given it unconsciously to others. Such a one I saw whose symptoms were those of so-called mysophobia. She dreaded touching any door handle, and never took off her gloves, and though they were dirty and so far dangerous, she still would not discard them.

From the moral effects of syphilis I now pass to the more material, and though I shall not trespass on the fields of pathology, yet I must preface my remarks by saying that I have to look to the toxic and general causation in which the blood is primarily affected, then to the characteristic affection of the vessels, and last to the more or less specific action on the nerve cells themselves. I have seen a few cases in which, unlike most nerve syphilitic affections the symptoms have arisen soon after the infection. In some of these cases the specific fever has been followed by marked disorder of the senses allied to delirium, and in some such cases progressive mental decay has followed; in others, where there has been parental syphilis, this has proved the starting of developmental general paralysis of the insane. I shall have to speak of the

former a little more fully later, but here I must say that I have met with a few cases in which the acute or early syphilitic affection has been followed by steady degeneration of the patient mentally and often morally. I have no post-mortem evidence as to what part of the brain is affected, but I presume it is the neurons and not the arteries, as is so well marked in the later forms of specific dementia.

As far as I can remember I have only once seen the febrile stage of syphilis followed by a distinct attack of mania.

In the monumental book on syphilis by Power and Murray, to which Mott contributes one volume, reference is made to neurasthenia following and being associated with syphilis, and I have elsewhere pointed out that the earlier symptoms of general paralysis of the insane may be neurasthenic, so that I need not here do more than record the fact that syphilis may start degeneration which may be manifest either through general neurasthenic symptoms or through evidences of progressive dementia.

The basis of definite delusions may be some sensory trouble. A deaf man may suspect others of talking about and against him, or may develop exaggerated opinions as to his personal importance because he does not hear contradictions.

I have met with several instances of specific neuritis in which delusions arose. There was a man connected with one of the largest racing stables who contracted syphilis. He had marked optic neuritis and blurred vision. He was in charge

of racehorses whose value, as he told me, was £40,000 or more. With the syphilis and imperfect vision he developed a not very unnatural belief that there were touts and spies watching his horses, and he made attacks on suspected but innocent persons and was sent for eye treatment to a general hospital, but there he assaulted the house-surgeon, suspecting him of being a spy.

In this case there was no suspicion of alcohol, as he said "If I had not been teetotal I should not have been trusted with these horses." In several cases I have suspected ear disease with being in part due to syphilis but I own I have no distinct proof of this, but I have seen cases with very marked delusions of suspicion arising from deafness.

In many ways allied to the above are the cases in which some facial disfigurement, such as loss of hair, of bones of nose, or marked scars about lips or face, have made the patient insanely self-conscious. I remember one man particularly who had a marked scar about his nose. He had borne this long, but with lack of occupation and the idea that this depended on his appearance, he was convinced that many people knew what the cause of his scar was, and hence they avoided him. In his case he tended to violence and had to be placed in an asylum for the safety of society.

I will next point out the association between fits and mental disorder, and their relation to syphilis.

That fits are not uncommon in cases of congenital

syphilis is recognized, and in the cases of the adolescent general paralysis of the insane they also occur, but most important is their appearance as the first symptom of general paralysis of the insane.

It is quite common for some slight attack resembling "petit mal" to occur in patients who have a history of syphilis, of old standing; such attacks as a rule are more transient than those connected with apoplectic attacks, but longer than those met with in ordinary Jacksonian epilepsy. In some cases I have seen a fit followed at once with evidence of some mental defect, and it has really issued in progressive dementia allied to general paralysis of the insane, but yet not following the ordinary course of this disease. I have seen such a case pass into a placid state of feeble-mindedness lasting for many years.

There is no true syphilitic epilepsy, but as I have just pointed out, there may be fits of slight or grave kind, due to syphilitic brain disease, which may either become habitual, but which as a rule tend to mental weakness much more rapidly than does simple epilepsy.

Associated with the above cases one has to recognize others with some such history as the following; contraction of syphilis in early youth, followed by normal active life, then some special strain, physical or moral, such for example as influenza on the one hand, and financial loss on the other, or war shock.

The strain is followed by a slight faintness, possibly with a very short period of unconscious-

ness. On recovery it is noted that there is some speech affection or some ptosis with external strabismus and pupillary affection.

Definite specific treatment is followed by apparent cure, but other and similar attacks may follow, leaving the patient more and more feeble in mind, or all the other symptoms of general paralysis of the insane may come on. In these cases there seems to be evidence of vascular changes leading to the local brain disorder.

It is such cases as these which lead to many mistakes as to the diagnosis of general paralysis of the insane.

Naturally you will expect me to say a good deal about general paralysis of the insane and its relationship to syphilis. First, then, I say that in my opinion, if not essentially a disease of syphilis it is generally due to toxins of which the spirochæte are in some way the most potent. It is always interesting to old men to think over and probably too often talk of their past experience, of the mistakes of others. I recall that in the early seventies the feeling was against syphilis being a cause of the disease. Men like Wiglesworth described cases of syphilis with general paralysis of the insane but did not see any true causal relationship, and I have letters from Sir C. Bucknill and from other leading authorities, opposing my contention that there was such causal relationship between the conditions. Early in 1888 I wrote on the arterial changes so constantly met with in the disease, and Crichton Browne and the workers in the West Riding of

Yorkshire asylum not only recorded this but did much to localize the sites of the adhesions of the membranes to the cortex, and to establish a connection between the cortical lesions and the mental symptoms. Yet it is only now that the true relationship between the two is being established by the monumental work of Mott. I must leave it to him and to others to show the exact connection, whether we are to look to the vascular or the neural changes as the primary ones associated with mental symptoms.

I shall not attempt to give any clinical picture of general paralysis of the insane, but I will rather give some of the results of my experience. First, you may ask, "have I ever seen a case of undoubted general paralysis of the insane recover?" I reply, "never recover, but patients may remain sane for many years." I have experience of men who, after the onset of the disease have had such complete remissions that they have resumed their business, or profession. I knew one naval man who brought a ship home from the East during a remission, only to die with characteristic fits a few weeks after his return to England, and I have known a clergyman conduct services for a few months before his final breakdown. I shall later have to say more about these remissions and their relationship to treatment.

Though there may not be complete mental recovery I have seen complete alteration in the mental state, so that the symptoms were those of chronic insanity, not of progressive mental decay.

Some twenty-five years since I saw a young man of considerable literary and artistic ability who, with the history of old syphilis developed all the most characteristic signs of the disease, and what was more, he went through all the recognized stages so that he became demented and had fits. Yet almost suddenly the whole picture was changed and he became a quiet, docile, feeble-minded man with no extravagant ideas, with no facial or lingual tremor. There were still changes in reflexes and pupils, but he now passes a quiet life and it seems as if he would live as long as he might expect even if he had had no syphilis.

Though rare, this case is not singular, and I have known another patient who, for 30 years lived a placid weak-minded life in an asylum where thirty years before he was supposed to be dying of convulsions due to general paralysis of the insane. In his case I may say there had been profuse suppuration, and I have seen other cases in which relief followed suppuration.

A question which used to be asked me was whether there was a disease, specific or not, answering to general paralysis of the insane but without any mental disorder. I have never known such, though I have in general hospitals seen patients in whom there were then no signs of mental disorder though the physical signs were manifest. For example, I saw many years since, with Dr. Bristowe in St. Thomas' Hospital, a man with all the physical signs of general paralysis of the insane but who was perfectly clear mentally.

I do not know why I suggested that he should have a seton in his neck, but he had, and he developed mental symptoms soon after.

I have definitely given my faith in syphilis as a cause of general paralysis of the insane, but I have also said that it alone is not efficient. Years ago I investigated what appeared to be racial differences. I had visits to Bethlem Hospital from Irish medical superintendents who wished to see cases of the disease because they thought in some way they must be overlooking them. They then came to the conclusion that the disease was very rare in the country districts in Ireland, though it occurred in the cities. The same was true in the Highlands of Scotland and in the rural parts of France. In America the Negroes as slaves very rarely developed general paralysis of the insane, but all these facts rather strengthen the specific origin of the disease, for syphilis is or was rare in the country places.

On the other hand, it has been said that in some of the wild parts of the world where civilization has meant the introduction of venereal disease and alcohol, little or no evidence of general paralysis of the insane is present. I fear I cannot give my authority but I recall a remark which I came across long ago that in Arabia there is a great deal of syphilis but no general paralysis of the insane. Maudsley used to teach that sexual excess was a cause of the disease, but he was writing before the rôle played by the spirochæte was known.

I am a follower of the work of Mott and from him

I gather that he has no doubt about there being a very distinct connection but at present he witholds judgment as to the exact physical or biological or biochemical connection.

The direct connection between syphilis and general paralysis of the insane has been on more than one occasion shown to me. Thus a husband contracted syphilis and later conveyed it to his wife. After many years he developed general paralysis of the insane and a year or two later his wife became a victim. I have seen brothers infected from the same source suffering from general paralysis of the insane.

The question of neurotic inheritance in general paralysis of the insane has often been considered, and in my experience general paralysis of the insane cases as a rule are less often neurotic by heredity than the majority of the other inhabitants of an asylum. I shall not go into the various forms of general paralysis of the insane which may be met with for I have no evidence that they bear any strict relationship to the syphilitic cause or its virulence.

In considering the relationship between general paralysis of the insane and syphilis I have already said that it is not often in my opinion the only cause, and I was much interested during influenza epidemics to meet with cases in which the symptoms of general paralysis of the insane only developed after the attack of influenza. It seems to me that there are many potential cases of general paralysis of the insane going about with a full sup-

ply of spirochætes hidden in their glands, yet no exciting cause may occur to start the specific degeneration. The war has produced many cases of men who, till exposed to strain or injury, showed no signs of brain degeneration. A shock, physical or moral, may be enough to awaken the organisms into activity.

Just as general paralysis of the insane has a distinct relationship with syphilis, so we now know has locomotor ataxy, and here I begin with the statement that a considerable number of general paralysis of the insane cases start with the ordinary ataxic symptoms and may be considered for a long time to be ordinary cases of that disease.

I shall therefore not refer to the cases of general paralysis of the insane which start with purely ataxic symptoms, but I have special interest in referring to ataxic patients who develop mental disorder of quite a different type from general paralysis of the insane.

In several cases I have seen insane interpretation of ataxic symptoms; thus, one man who had marked joint affection as well as some skin eruptions, ascribed the whole to special influence of the devil who had him in possession. But beside I have seen several cases of locomotor ataxy with very marked mental depression, true melancholia, with suicidal tendencies, but the local pains and sleeplessness might be almost enough to account for the mental depression. The more interesting cases were examples of the insane interpretation of morbid sensations due to locomotor

ataxy. Thus one man with gastric crises accused people of giving him poison, another with rectal crises had the very insane notion that an enemy was able to introduce a cat into his rectum and then pull it out, the cat resisting, causing his agony. The most important thing to recognize is that whereas cases of general paralysis of the insane live only a few years, the insane ataxic may live for very many years with no further evidence of mental degeneration.

What has been called juvenile or adolescent general paralysis of the insane is not very common, and I believe the reason or one reason is that most of those who might develop the disease die in earlier years of convulsions. In all the cases which I have met with there has been parental syphilis, and the disease may pass rapidly into dementia with or without fits. A certain number of these patients are sent to institutions for idiots and imbeciles.

As I am not to-day demonstrating the symptoms which may be present in the various forms of mental disorder, but only their relationship to syphilis, I need say no more on this subject but pass on to the very important question as to the part played by inherited syphilis in the production of permanent mental weakness, imbecility and idiocy.

Up to quite recently it was generally said that few idiots owed their defect to this disease, but that was before the Wassermann test was known and when the physician depended on the more marked

physical signs of the constitutional disease, and here I may say that I have seen very few cases in Earlswood in which the more obvious signs of constitutional disease were present. More recently there have been statistics from idiot asylums which have altered one's judgment and at Earlswood a careful examination of the blood gave positive reactions to the Wassermann test in a very large proportion of the patients examined, and I may say the examination was carried out at a general hospital laboratory and the patients were not selected. It is well to recognize that in most of these cases the parent, who transmitted the constitutional disease, showed no signs of it himself.

As we all recognize the cases of brain syphilis occur more frequently in patients who have had very slight symptoms in the earlier stages of the disease.

A large mass of evidence is being collected in America on the forms and causes of degeneracy, and all seem to make it clear that among defectives, whether moral or intellectual, syphilis is a partial cause. I have been struck by morbid heredity; I remember one case in which several children were born before the acquisition of syphilis, and these children grew to maturity normal, while children begotten after were defectives. In the same way I have seen a fair number of children of a general paralysis of the insane parent who having been begotten before the active mental disorder was present, were normal, while children begotten by a definite general paralytic stand a poor chance. In

these cases the constitutional syphilis in the parent was in abeyance till the more active life of the spirochætes arose.

It is always difficult to separate the family from the physical environment. Thus I have been consulted about abnormal children whose home life and parental care were bad, and I have met a few cases in which with a licentious parent there has been a child with the most terrible precocious sexual depravity. The moral defects associated with constitutional syphilis are innumerable. I remember one girl of 6 who was given to masturbation, and openly said she hoped to grow up to teach other little girls her habits.

On this point, then, I have to sum up that constitutional syphilis which may not have been evident in a parent, may give rise to almost every form of mental degeneracy in children.

As I have already said, a generation ago those connected with idiot asylums were agreed in stating that the obvious evidences of constitutional syphilis to be observed among their patients, were very few, in fact that less than 5 per cent. of the idiots had any outward signs of the constitutional disease. Now with the Wassermann reaction the whole opinion has changed, in fact, so radical has been the change that I have—with Mott, wondered if it was all true. Anyway Drs. Fraser and Walker reported after very careful observation, that in their experience 50 per cent. of mentally defectives show positive reaction, that constitutional syphilis was a main cause of epilepsy as met with in men-

tally defectives. They had to admit that there were many children apparently normal who gave a positive reaction, and last they agreed with what I have said that in most of these cases there were neither Hutchinson's teeth nor specific iritis.

All this is most important, though I fear we cannot say that we know how to meet the potential mental weakness before it shows itself, for we cannot examine every school child for syphilis, and if we did we do not know how to meet the constitutional enemy.

We have to guard against being carried away by the fashion of the day. Every new fact in pathology is eagerly seized by the workers in medicine and is tried to be fitted into every possible condition. We are as subject to waves of thought as ever men were. Bacilli may reign to-day but humors, toxins or vaccines will rule to-morrow. Every wave leaves something behind, it may only have destroyed old landmarks but it will leave a shore-line of its own. The syphilitic interpretation of every nervous disorder represented the high tide and now I think we have gained a shore-line of experience which is nearer the truth.

And now, gentlemen, I have to leave the subject, pointing out that I have endeavoured rather to be a directing post than anything else. I would once more appeal to my tables and emphasize my belief that though syphilis may have had a very potent weight in the production of the disorders yet it is rarely the only cause.

THE PROGNOSIS OF EXOPHTHALMIC GOITRE.

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It frequently happens that medical writers claim good results from a particular treatment of a disease without contrasting their findings with the course of the disease when the treatment they advocate is not given. If the natural tendency is that 75 per cent. of the sufferers from the disease in question get well if just left to themselves, anyone giving, say, peppermint water would find that he had 75 per cent. of cures, and might conclude that the peppermint water was beneficial, which would be erroneous. It cannot be that we can gauge whether treatment is helpful until we know the course of a disease if left to itself, or, in other words, prognosis. That is essentially what patients need; their only concern is whether or not they will recover. If they do, it matters little to them what name we give to their disease or whether we adopt any treatment. Sometimes the outlook is certain. For example, a sufferer from acute suppurative peritonitis will almost certainly die if left to himself, so will a man

afflicted with cancer of the liver. Failure to study the natural course of a disease has led to all sorts of unnecessary treatment. For example, before it was discovered that salicylates shorten rheumatic fever and diminished its complications, various drugs were given that had no effect in shortening the disease or lessening its complications, as was demonstrated by Sir William Gull in his famous paper on the treatment of rheumatic fever by peppermint water—that is to say, without any treatment. His cases did just as well as did those treated by any other method then in vogue, showing that the treatments then known had no effect for good on the disease. Again, good results have been claimed for all sorts of drugs given for chorea, but those who claim these results are forgetful of the fact that the disease gets well just as fast if no drugs are given. This morning I propose to relate to you the facts ascertained by following the after-history of cases of exophthalmic goitre, in order that then you may be in a position to estimate the value of any treatment you may adopt.

From 1888 to 1907 there were admitted into Guy's Hospital 169 cases of exophthalmic goitre; of these twenty-one died. Letters were written to all the remainder and fifty-four could be traced. From 1894 to 1909 I saw in private practice fifty-seven cases of the disease. Those who had undergone operation for the disease were excluded from both sets of cases, as the desire was to study the disease if left to itself. Doing this left me with

forty-nine hospital and fifty-three private patients who could be traced. Some of these I interviewed, in the others either by letters from doctors or the patients themselves their history was obtained. The investigation was undertaken in 1910, and then among the forty-nine hospital patients eight were dead, and among the fifty-three private patients seven were dead. With the help of my friend, Mr. H. C. Thiselton, an actuary, a comparison was made of the actual deaths with the expected deaths according to the "healthy females experience of twenty British offices amongst assured lives." This showed that while among the hospital cases eight died, the expected deaths among a like number of similar healthy women would have been five, and that among the private cases, while seven died, the expected deaths among a like number of similar healthy women would have been three. The figures here dealt with are for actuarial purposes, too few for precise results, but the general conclusion may be drawn that the mortality experience among sufferers from exophthalmic goitre is heavier than would be expected according to a well-known standard table. The total deaths were fifteen against eight expected. These figures suggest that if it were possible to obtain a great many more cases we should probably be able to prove that the expected mortality of sufferers from exophthalmic goitre not operated upon who have been in a hospital and discharged, and of sufferers from it in private practice who sought a second

opinion (for all the private cases were referred to me by their usual medical attendant) would be about twice as great as among healthy women. But it must be remembered that nearly all the patients were of such an age that the expected deaths among the healthy are few, and therefore twice the expected number is not a great number. But again this suggestion must not be transferred without qualification to exophthalmic goitre generally, because all the patients here considered were ill enough either to come into a hospital or desire a second opinion. The inclusion of all cases of exophthalmic goitre, and therefore of many slight cases, would almost certainly make the expectancy of life better than appears from these figures. On the other hand, inclusion of those who died in the hospital would make the mortality higher than appears; but ninety-four patients who were well enough to leave the hospital cannot be traced, yet if we could include them we should be able to some extent to balance the higher mortality which would result if we included those who died in the hospital.

Of the twenty-one who died in the hospital three died as a result of operations on the thyroid, leaving eighteen. It is well known that diarrhœa, and to a less extent vomiting, often accompany exophthalmic goitre. One or both of these symptoms was very evident in eleven of the eighteen. These figures indicate that it behoves us at once to put the patient to bed when these symptoms supervene. Often there is naked-eye evidence of

gastro-enteritis at a post-mortem examination. Six of the eighteen became delirious and one became comatose. Two had melancholia, one had mania. Two had endocarditis, two had rheumatic fever, and one attributed the exophthalmic goitre to this disease. These cases exemplify the well-known association between the two diseases. One died of diabetes. Of seven private patients who died apart from operation, one succumbed to phthisis, one to pleural effusion, one to heart disease, one to alcoholism, one to diarrhœa, and two to diabetes. Deaths from diabetes are of interest when we remember that glycosuria is a common symptom of exophthalmic goitre; the deaths from delirium, coma, melancholia and mania serve to remind us of the recognized association between exophthalmic goitre and mental disorders.

We will now see what is to be learnt from the cases which were traced and did not die. If we exclude those which were operated on and one in which the information was too scanty to be of use we have forty left. Of these twenty-six have done very well, twelve have done well and describe themselves as better, and two are not well. With regard to those that have done very well, No. 1 has required no treatment since she left the hospital twenty-one years ago; No. 4 was a bad case in the hospital twenty years ago, has worked hard for eighteen years, has married, has three children, and is now a widow; No. 6 in the hospital twenty years ago has worked since then as a district nurse, subsequently married, and has

four children; No. 9 in the hospital sixteen years ago feels quite well, has not had a doctor for twelve years; No. 15, when in the hospital eleven years ago, had had the disease for ten years; after leaving the hospital, since when she has had no treatment, she has married and has six children; No. 18, in the hospital eight and a half years ago, is now quite well, and has been in service for five years without a single day's illness; No. 24 is able to do work; No. 25 is working as a cook; No. 29 does not know that there is anything the matter; No. 32 is now quite well and has been so since her discharge; No. 34 is wonderfully better; No. 35 is perfectly well; No. 37 has had no return of her old complaint and is married; No. 40 was in the hospital eight and a quarter years ago, has been well ever since, went into service for four years, then married, has four children, and never needs a doctor, "although she pays into a club"; No. 47 was quite cured by an American goitre cure; No. 48 attributes her improvement to pregnancy. I need not weary you with any more descriptions; all these twenty-six cases have done as well as those sixteen just briefly quoted.

Turning to the group of twelve whom I have classified as having done well, the following may be quoted as examples of their present condition: No. 11, although she had several attacks of mental derangement, is considerably better and in a situation; No. 13 says her health is fairly good, she took a situation after leaving Guy's, then she married, and she has two children. All the others

of this group say they are much better, and no doubt some might with justice have been placed in the previous group, and even the two that I have, from the letters they wrote to me, classified as "not well" say they are better than when they left the hospital. It seems therefore to be a fair conclusion to say that these forty hospital cases have done well, and that judging from them exophthalmic goitre appears likely to get well even if no operation is performed, a conclusion borne out by our clinical experience, for we do not often see elderly women suffering from it, and the mortality is certainly not severe enough to account for this.

Now let us consider the private cases, fifty-seven in number. Of these some died, some were operated on. Of one the information was too scanty to be of use, so that in all there are forty-seven left to be considered. I have grouped them in the same way as the hospital cases, and find that thirty-five have done very well, nine have done well, and three are not well.

With regard to the first group, as with the similar group of hospital cases, they have done very well. No. 4, a severe case, although very nervous, has done very well and is still improving; No. 6 is of great interest, for she was very ill when I saw her, but now she does her household duties and goes for long walks; No. 7 was also very ill, but her pulse when her doctor wrote to me was 80, and she was expecting her confinement daily; No. 8 is quite well; No. 9 is working as a school mistress; 10, 12 and 14 are quite well; 15 takes

average amount of exercise and is not debarred from any social function; 17 was very ill indeed, I have never seen greater exophthalmos; it is now much less, and she can walk three miles; 18 is completely cured; 20 in seven months gained 28 lb.; 21 is of great interest, for seven years before I saw her she had had one lobe of the thyroid removed for the disease, she got better for a time, but the disease returned, and when I saw her she had most of the symptoms of exophthalmic goitre present in an extreme degree, and the remaining lobe of the gland was enormous, but after prolonged continuous rest she got quite well, and can now walk to business and do a hard day's work; her sister had the disease severely at the time the patient underwent the operation, she was not operated on, I saw her at the same time as her sister was ill and she was well; 22, 23, 24, 25 and 26 call for no comment, they are all in good health and apparently cured; 29 is in very good health and goes to dances; 30 also says she is in very good health and busy from 7 a.m. to 10 p.m.; 31 did very well; 34 was very ill, she had a loud systolic murmur and great œdema of the legs, yet "she got practically well and went to New Zealand"; 35 does not call for comment; 36 can go for long walks; 39, a very severe case, got quite well; 40 got quite well; 41 was a very severe acute case following influenza, the thyroid became much enlarged in fourteen days, and when I saw him the restlessness and sleeplessness were so formidable and his pulse

was so weak that it was thought he would die, yet within twelve months he was riding across country; 42 got quite well; 44, also a severe case following influenza, got quite well; 45 occurs in the list of those who died, but she got well of her exophthalmic goitre, and then some years later fatal diabetes appeared; 48 many years ago had exophthalmic goitre and a fibroid of the uterus, when the fibroid shrank so did the thyroid, and she got well (except for slight exophthalmos) of her exophthalmic goitre; I saw her for acute pneumonia, from which she recovered at the age of 57 without any return of the exophthalmic goitre; 49 was of interest as being a very severe case; No. 50 was a severe case, when I first saw her, fourteen years before, she had glycosuria; this disappeared and has not returned; six years after she was first seen she could walk eight miles, and she has for many years been perfectly well; in No. 51, a very severe case, the exophthalmic goitre was attributed to influenza, a loud systolic murmur was audible; he had much œdema of the legs and he got perfectly well; Nos. 55 and 56 got quite well.

There is not much to say about those nine in number who are classified as having done well. Some of them had other diseases which would explain why they had not done better, others would not rest as advised. Three have not done well, but none of them would rest as advised.

Putting together the forty hospital and forty-

seven private patients we have considered we get :—

	Done very well	Done well	Not better
40 Hospital cases ...	26	12	2
47 Private cases ...	35	9	3
87 Total	61	21	5

Therefore the private cases have done somewhat better than the hospital cases, but really much better than these figures show because the mortality is higher among the hospital than the private cases. I arranged the cases into three classes, mild, severe and very severe, and found that more private than hospital cases in the severe and very severe classes did well. This supports the conclusion that the outlook is better, the better the social position.

Naturally you will ask about the treatment of these cases. The first thing to notice is that several patients stayed quite a long while in the hospital (one was in 170 days, another 95 days) and yet did not improve during their stay, yet they ultimately got quite well or nearly well some time after they left the hospital, and when I wrote to them they had remained well, although the mode of life of hospital patients outside the hospital is the reverse of what might be expected to aid a cure. Therefore we must conclude that some sufferers from exophthalmic goitre may recover without any treatment.

The treatments used in these series of cases have been so various that it is impossible to draw any conclusions about any of them, but from my own

experience and from what I have read, my strong impression is that the most important thing is rest in bed for weeks and months together with freedom from anxiety. A woman came into the hospital suffering from exophthalmic goitre; while in the hospital she had an attack of typhoid fever, her exophthalmic goitre gradually improved, and she got quite well of it. Most likely the long rest compelled by the typhoid fever was the cause of the cure of her exophthalmic goitre. Probably it is because private patients are better able to rest than hospital patients that the outlook for them is better. Next to rest comes food; no patients do well if they lose weight, and those who are thin will not improve until by abundant food they gain weight. If they are very nervous, excitable, or sleepless, it is necessary they should be quieted and given sleep. For this bromides often suffice, but it may be necessary to use hyoscine. If the pulse is very rapid it is probably wise to give digitalis. Beyond the benefit of such symptomatic treatment there is no evidence that drugs are of any use. Many of the women who are cured bore children; this does not do them any harm, and they stand confinements well. Patients who are once cured do not often relapse, hence readmissions are few.

During the last few years many cases have been operated upon, and later on we shall have had enough experience of this treatment to contrast the results with those here given, and then I trust a numerous series of cases which have been followed

for some years after operation will be published, we shall then see whether operation lowers the mortality and shortens the course of the disease. We all hope it will do both.

This is not the occasion to deal in any detail with the operation, but we must remember that it must not be lightly undertaken. Patients with exophthalmic goitre require skilled anæsthetists and skilled surgeons. I have known them die from the anæsthetic and as a result of the operation. Once I saw a case, already mentioned, in which half the thyroid had been removed; not long after the remaining lobe began again to become large, and the patient had a second and very serious attack of exophthalmic goitre, from which, under rest, she recovered with difficulty.

GRAVE FAMILIAL JAUNDICE OF THE NEWLY BORN.

By Sir HUMPHRY ROLLESTON, K.C.B., M.D., F.R.C.P.

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Brief Description.

In grave familial jaundice, successive infants become jaundiced a few hours to a day after birth, pass into a drowsy condition, and usually die within a week, often with convulsions; it resembles physiological jaundice in its onset, and, like it, does not show any gross post-mortem appearances to account for the jaundice.

Pathogeny.

Pfannenstiel regarded it as only an intensive form of the physiological jaundice, but not only is the prognosis very different, but there may be evidence of maternal toxæmia during the pregnancy. In three (Nason, Tylecote, Rolleston) out of the twenty-five family groups that I have collected—that is, in fifteen out of 130 cases of newly-born infants with grave familial jaundice—the mothers had recurrent jaundice during their

pregnancies. In the families reported by Smith and by Ritchie, the mothers had jaundice at the birth of one of the children, and in Arkwright's family of fourteen cases with four recoveries the mother had had jaundice when four years old and was always "sallow." In Blomfield's cases, the mother suffered much from severe vomiting during the pregnancies, but this association appears to be exceptional. Against the view that this grave disease is due to maternal toxæmia, and that in its most complete form the mother is jaundiced as well as the child, it might be argued that, as a rule, the infants suffer much more than the mothers and that the jaundice does not appear until after birth; but it is obvious that similar objections might be raised to the transmission of congenital syphilis; and it may be added that in congenital obliteration of the bile ducts there is a similar or greater delay in the onset of jaundice. The following observation is perhaps in favour of the existence of maternal toxæmia:—

I gave the mother, who had had recurrent jaundice in three successive pregnancies with fatal familial jaundice in the infants, hexamine and salicylate of sodium in her fourth pregnancy; in the eighth month, she became somewhat jaundiced just before the birth of a boy who was never icteric and was well ten years later. During her fifth pregnancy she was treated by Dr. Spencer Lewis with small doses of hydrargyrum cum cretâ but was jaundiced during the eighth and ninth months of pregnancy, and bore a girl who was slightly jaundiced and died of acute summer gastro-enteritis when seven weeks old.

Benedict's cases of recurrent jaundice, with pruritus and hepatic enlargement, in two sisters

during their pregnancies, these manifestations passing off after abortion or premature delivery and the offspring escaping icterus, might perhaps be regarded as incomplete cases and corresponding in this respect with the cases in which the offspring alone are jaundiced. It is highly desirable that the metabolism of the mothers of infants with familial jaundice should be thoroughly investigated, especially during their pregnancies. In the meanwhile, it is suggested that *in its most complete form the disease is hereditary, the mothers becoming jaundiced during the pregnancies of jaundiced infants, and that the usual familial form is an incomplete manifestation of the morbid processes.*

Ætiology.

Some of the families, in which the disease has been reported, are remarkable for the large number of pregnancies; in Arkwright's there were 15; in Auden's, 12; in Underwood's and Ritchie's, 11; and in Busfield's, Nason's, and Morris's, 10. Occasionally there are some miscarriages or premature births, but all the eleven pregnancies in the family reported by Ritchie went to full term. Some writers refer to the robust condition of the infants; but all Arkwright's cases were rather weakly, and one had an intra-uterine amputation of the forearm.

The disease appears to be less likely to attack the first and second born than the later infants in the families affected. The first-born escaped in the families reported by Arkwright, Smith, Auden,

Busfield, McGibbon, Morris, Tylecote, Still (two families), and the first two children in those reported by Abt (two families), Duguid, Nason, and Ashby and Wright. The first-born therefore escaped in fourteen out of the twenty-five families collected, and in several families the first-born was slightly jaundiced and was the only one to recover. There is no evidence that syphilis plays any part in the disease, and though infection has been put forward as a cause, there is no good reason to accept this.

The sex is not mentioned in some of the reports, but in those that I have collected there were thirty-one males and thirty-one females. The sex incidence thus differs from that of congenital hypertrophy of the pylorus and of congenital obliteration of the bile ducts, which are both, especially the former, much commoner in the male sex. It has occurred in twins (Still).

Morbid Anatomy.

There is bile-staining of most of the tissues, and, as in physiological jaundice, there is selective staining of the lenticular and other nuclei in the brain, while the cortex escapes. Punctate hæmorrhages in the viscera are described, but their importance is doubtful, for they are not uncommon in stillborn children. There is no gross obstruction of the bile-ducts in the recorded cases except in one of McGibbon's cases, described in detail by Buchan and Cowrie, in which there was obstruction but not obliteration of the biliary papilla.

Clinical Features.

Afebrile jaundice usually appears a few hours to a day or two after birth, and increases; but, as in other forms of jaundice, may show curious variations. The infants may be born icteric; Smith describes a jaundiced stillborn child, and noticed in his three cases that the liquor amnii and membranes were bile-stained. The stools are not acholic; the urine may or may not contain bile. Hæmorrhages are not a feature of the disease. In a few instances enlargement of the liver and spleen has been present. The infant becomes drowsy, and usually death occurs within the first fourteen days, often during the first week, and commonly with convulsions. Recovery after prolonged drowsiness appears to be more likely to occur in the earlier infants of the family; in nearly all the reported cases of this familial jaundice, recovery, when it occurs, has been complete; but one of Morris's cases, a girl, survived for four and a half years, and was jaundiced practically all the time.

Prognosis.

This is bad; out of 130 collected cases 100, or 77 per cent., proved fatal. It is possible that prophylactic treatment of the pregnant mother (*vide* p. 38) may improve the outlook in the future. Spiller has recorded four cases of spastic diplegia following recovery from severe jaundice in the newly-born, but none of these was stated to be of the grave familial form. In connection with this and with the selective staining ("kernicterus") of the cerebral nuclei in grave familial and in physio-

logical jaundice of the newly-born, it is interesting to recall Wilson's comparison with the selective action of a poison, probably non-microbic and possibly a lipoid, causing bilateral degeneration of the lenticular nucleus in the disease progressive lenticular degeneration, which is constantly associated with hepatic cirrhosis of the portal, not of the biliary, type and is often familial.

Diagnosis.

Reference may be made here to the other forms of jaundice in the newly-born.

Simple, Physiological, or Idiopathic Jaundice has been estimated to occur in from 33 per cent. upwards of all births. Breschet believes that it is constant, and Bang found that the blood in the umbilical vein always contains bilirubin and that the amount of bile in the blood of the newly-born increases up to the third day of life, after which it begins to diminish. The jaundice begins within the first two days, and appears earlier than in the cases of grave infective jaundice. There are no symptoms, though sleepiness is mentioned by Langmead. No treatment is necessary, and the main points of interest about these cases are (*a*) in the first place, the differentiation at an early stage from the rare condition of grave familial jaundice, which depends on the history of the family, and (*b*) in the second place, the causation.

Congenital Obliteration of the Bile Ducts is accompanied by hepatic and splenic enlargement, and practically always proves fatal within eight months; it is, therefore, relatively chronic as com-

pared with grave familial jaundice. It is not a familial disease, but the first case of grave familial jaundice in a family would be very difficult to distinguish during life from one of congenital obliteration of the ducts; hepatic and splenic enlargement and acholic stools would be in favour of gross obstruction. The pathogeny of congenital obliteration of the bile ducts has been much discussed. It is generally agreed that it is not due to syphilis, though there are a few cases (Beck, Simonini, Rolleston) of congenital syphilitic stricture of the common bile duct on record. John Thomson, Milne, and Lavenson consider that there is in the first instance a developmental aplasia or narrowing of the ducts which gives rise to obstructive cirrhosis. My own view is that a maternal toxæmia causes cirrhosis followed by a descending obliterative cholangitis; and Dr. S. Wyard suggests that there is an ascending cholangitis from the duodenum set up by the irritation exerted by the swallowed liquor amnii. Most writers agree that all cases are not necessarily due to the same mechanism.

The Severe Infective Forms of Jaundice, such as those due to umbilical, cutaneous, or intestinal infection, formerly occurred in epidemics, and were responsible in the old insanitary lying-in hospitals for a terrible mortality, but, as far as I know, are rare now. The jaundice comes on about the fifth day of life, or later than in familial jaundice, and is accompanied by signs of grave septicæmia, high fever, and hæmorrhages especially from the umbilicus or the alimentary canal.

Winckel's disease (epidemic hæmoglobinuria with bronzing of the skin) and Buhl's disease (acute fatty degeneration) are manifestations of this septicæmia of the newly-born, the infection probably entering from the intestines. The prognosis is very grave. Treatment should consist in the administration of anti-streptococcic serum, washing out the bowel, and the oral administration of intestinal antiseptics such as minute doses of calomel.

In Melæna Neonatorum jaundice is often present; thus Pitfield records as hæmophilia four successive males with jaundice and hæmorrhages.

Congenital Splenomegalic Hæmolytic Jaundice runs a mild course, and can hardly be called a disease; it is recognized by the characteristic fragility of the red blood corpuscles when tested with hypotonic solutions of salt. *Congenital syphilis* does not often cause jaundice; it usually comes on early and is accompanied by other signs of the disease such as enlargement of the liver and spleen. Secondary pyogenic infection may occur in these cases.

Treatment.—On the hypothesis that grave familial jaundice of the newly-born is due to fœtal toxæmia of maternal origin, the prophylaxis should consist in care of the mother's diet and the administration of intestinal and biliary antiseptics, such as hexamine, salicylate of sodium, minute doses ($\frac{1}{20}$ gr.) of calomel, tetrachloride of naphthaline, salol, and guaiacol. Ballantyne recommends chloride of calcium.

The infant should be treated with small doses

of calomel, which some authors consider to have done good, and should not be given the breast.

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THE VALUE OF COMBINED TREATMENT, WITH SPECIAL REFERENCE TO SURGERY, ELECTRICITY, AND X-RAYS.

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Gentlemen,—I would recall to your minds for a moment the story of Naaman the Syrian. He was a leper, and went to consult the Prophet Elisha as to what he should do to be healed. The advice given was that he should wash seven times in Jordan. But upon this Naaman fell into a great rage, his indignation focusing itself, as it were, upon two points; he objected to the bathing place as such, but more particularly and strenuously he quarrelled with the simplicity of the prescription. And he turned and went away in a rage. But his servants, displaying both insight and courage, said to him, "My father, if the prophet had bid thee do some great thing, would'st thou not have done it?" And Naaman—so we are told in the fifth chapter of the Second Book of Kings—took to heart the gentle reproof, washed seven times in Jordan, and was healed.

The story, gentlemen, is not a digression, or

in any sense irrelevant to my subject. For this medical generation to which we belong is too apt to seek always to do some great thing, to look for a sign from heaven—which shall not be given unto them—and, incidentally, to stone the prophets. Probably none of us are altogether guiltless in this respect, but it is a pleasant exercise to point out the failings of others, and I venture to say that some surgeons, at least, in the presence of certain serious maladies, spend their time in sighing for serological signs and wonders, instead of utilizing means which, if they do not accomplish results with dramatic suddenness, are, at any rate, of proved value. The septic wound which persistently remains septic despite thorough surgical cleansing and drainage, and which threatens to kill the patient by exhaustion, is a case in point. Too often one hears the pious hope expressed that in the future a serum will be discovered that will neutralize all wound toxins; or, better still, some chemio-therapeutic preparation which when injected, will kill all the pus-forming organisms. The discovery of such agents is a legitimate object of research; but, meanwhile, the aid which can be given by light, X-rays and various forms of electricity should not be neglected.

The value of these agents in the treatment of septic wounds can no longer be doubted. The proofs are set forth in numerous monographs published during the last few years, and the whole subject was discussed at length at a meeting of the Electro-therapeutic Section of the Royal Society

of Medicine last year. Equally, I shall ask you to take for granted beneficial effects of X-rays in localized tubercular conditions, and in malignant disease; also their power to disperse pathological fibrous tissue. In short, for the purposes of this lecture it will be assumed, on the one hand, that most of the statements made by responsible practitioners of electrical medicine are justified; and, on the other, that modern surgery can accomplish all, or nearly all, that its exponents claim for it.

What I want to put before you is that, in many instances, no single method of treatment is adequate to cure a patient—or, at any rate, to cure him with the minimum of risk, pain, and delay.

Let us consider first the question of tubercle. Tuberculous affections of the skin are notoriously difficult to deal with. It is but rarely that surgical scraping in itself leads to an ultimately satisfactory result. The operation is performed, an apparently granulating surface is obtained, and all goes well for a few days. Then, too often, the healing process becomes arrested, and, to a greater or less degree, the disease recurs. The outcome of unaided treatment by X-rays or light is not seldom equally disappointing. But a combination of surgical and electrical methods, while not infallible, gives much better results. On the surgical side, it is necessary to curette almost as deeply as if one intended to depend on this alone, and also to go at least a quarter of an inch beyond the margins of the diseased patch. The wound

will, of course, begin to heal from the edge in the normal way, leaving a healthy granulating surface which decreases in size day by day. The object of post-operative treatment by electricity and radiation is to keep this process going. It is not easy to describe in words the appearance which must be maintained, but anyone familiar with the healing of granulating wounds can tell by daily inspection whether or not healing is proceeding normally.

To secure such orderly progression is not by any means always a simple matter. Sometimes the daily application of a very small dose of X-rays is all that is necessary; but more often one finds that after a short time a particular form of stimulus fails to act, and the wound hangs fire; or, what is worse shows a tendency to false healing. It is here that skill and experience come in; but it may be said briefly that the secret of success lies in variation of stimuli according to the behaviour of the wound. Short sparks from a high frequency vacuum electrode and zinc ionization suffice as variants in most cases. Sometimes it is beneficial to cause a sharp reaction by ultra-violet rays from a tungsten arc; or it may even be necessary to resort to further curettage of a part of the diseased area. An essential point is that not the smallest scar must be left at any point; for if such remains, it is nearly always an indication of unsound closing, and the probability of recurrence is great.

Once genuinely healed, a particular patch is not likely again to become the seat of tubercle; but,

of course the constitutional tendency remains, and should be combated by all means.

Whether or not tuberculin in any of its multitudinous forms should be numbered among these means may be left to serological experts to determine, but I take the opportunity of emphasizing thus early that the advocate of true combined treatment cannot concern himself with local measures alone, but is bound to call in the aid of any vaccine, serum, or drug which is of *proved* value in combating the morbid condition with which he is dealing.

In some cases, for example in the local lesions of syphilis, there is little likelihood of this principle being neglected. But we must always remember that there is a constitutional factor in most local disturbances. When these troubles are the result of bacterial invasion, lip-service, at any rate, is paid to this truth; but it is scarcely so well recognized that even in the case of traumatic lesion of an aseptic nature, one must beware of not seeing the wood because of the trees. For example, a simple fracture may be slow in uniting chiefly because of thyroid deficiency, and will be speeded up by administering the appropriate extract.

Tuberculous neck glands offer a useful field for combined surgical and electrical measures. Surgery alone often fails to eradicate the trouble, and its cosmetic results are sometimes far from satisfactory. Recently, for example, I saw a child of 12 who had had 14 operations on her neck in 6 years, and I know of many cases in which four or

five excisions or scrapings have been undertaken within a comparatively short space of time.

This is certainly not a desirable state of affairs, and in an endeavour to better it, attempts were made years ago to cure tuberculous neck glands by X-rays. Some few attempts succeeded, and were reported in medical journals; but mostly what happened was that after some weeks of rapid improvement progress became arrested, and eventually recurrence took place.

X-ray treatment, therefore, fell into disrepute, but unjustly so. Used for certain definite ends it is invaluable.

The rule to follow may be briefly stated as follows: When there is no detectable softening, X-rays should precede surgical treatment by six or eight weeks, small doses being given three times a week. This causes all surrounding inflammation to subside, and the gland itself usually shrinks until it can be detected only by careful palpation. This is the time to operate—only a small incision is required, and the gland can be readily excised. If the surgeon decides to close the wound at once, no further radiation treatment need be given, but if there is any subsequent discharge, a few post-operative sittings are desirable.

If on the other hand, softening has already occurred when the case is first seen, no X-ray treatment should be given before operation. The resulting sinus, if left to itself, may discharge for months, but X-ray treatment commenced within a day or two will generally result in healing, with a minimum of scar tissue, after a few weeks.

The surgeon on his part, must, of course, see to the vital matters of free drainage and healing from the depth no less carefully than if X-rays were not being employed. It may be laid down here as a general proposition that if combined treatment is to succeed, its various component measures must be carried out as fully and skilfully as each would be were it alone being expected to effect a cure.

I come now to the question of malignant disease. X-rays are of greater or less value in most neoplastic conditions, but they have their most striking success in cancer of the breast, and to this I purpose confining my remarks. Malignant growths in this region can be made to disappear by X-rays alone, so far as inspections and casual palpation are concerned; but rarely to such an extent that remains cannot be detected when carefully sought for. Far more important is the fact, long suspected on clinical grounds, but recently experimentally proved, that X-rays in suitable doses raise the immunity of the body to cancerous invasion.

Radiotherapy should therefore be administered before operative removal. Such pre-operative treatment has the dual advantage of depressing the cancer cells locally before any surgical trauma has disturbed them; and, at the same time, increasing the general powers of resistance to any subsequent migration.

In view of what Sampson Handley has shown as to the radiating growth of cancer along the

lymphatics, it is obvious that local treament should embrace a very wide area. The individual doses should be small, and repeated two or three times a week; it is possible to obtain a quick local effect at the expense of the general resistance. From two to three weeks should be spent in this preparatory treatment.

Post-operative raying is, of course, a well-known procedure, and it should at this date require no argument in its support. Nevertheless, there are still many surgeons who do not advise it, or do so only when recurrence is already manifest. It is true that excessive radiation may do harm by lowering resistance; but experience has taught us the limits of safety.

No one disputes the value of prophylactic inoculation because of the harmful effects of over-dosage. In my opinion, the best form of post-operative raying consists in giving a course of about a dozen sittings spread over a month or six weeks, with periods of rest of two to four months between. This should be continued for at least two years. If this treatment be carried out properly, it can be practically guaranteed that the patient will have no externally visible recurrence.

The case which is, at first sight, surgically inoperable—or, at any rate, incurable—offers much scope for a combination of surgical and electrical treatment. Fortunately, one does not in these days very often see hopelessly advanced cases, but if there is already a fungating mass, it may be destroyed by the diathermic cautery, and the

raw surface treated by X-rays. By these means one can sometimes bring about surface healing, and reduce in size and render less adherent the main growth, so that all or most of it may be removed surgically, and a satisfactory external appearance results. Of course, in such cases the disease has nearly always reached the chest cavity. But even here X-rays are not powerless, and such patients may live in comfort for months, occasionally for years. Only when the cancerous process has reached the spinal bones does the case seem beyond the possibility of any relief—at least, so far as our present experience goes.

I have purposely deferred until now to refer to septic wounds, as they are so firmly rooted in the professional mind as almost wholly the products of war. But, so long as industrial accidents occur, it is unlikely that we shall completely banish the septic wound—and its sequela, the septic amputation stump. A seriously infected stump after an amputation—say, below the knee—may mean the loss of the entire limb, if not the death of the patient. In the early stage, characterized by acute inflammation, profuse discharge, and severe pain, there is nothing so useful as the incandescent lamp of 2-3,000 candle-power. Physicists tell us that ordinary light rays alone are produced in such a lamp; nevertheless, experience teaches that its glass should be of a kind readily permeable by rays of short-wave length. The effect of the combined light and heat rays is to cause the raw surface exposed to them to ooze serum freely,

while at the same time pain is relieved. After a few days, when the acute stage has subsided, zinc ionization and X-rays should be applied to the stump on alternate days. Surgical drainage and irrigation of dead spaces is likewise necessary, but by combination of means re-amputation may be avoided in many cases in which at first sight it appears inevitable.

The good effects of X-rays in causing the absorption of keloid are well-known, and electrical departments during the war were overwhelmed with requests to deal with exuberant and unhealthy scar tissue. But the sad part was that in many, if not in most, of these cases the deforming scars need never have been allowed to form. It may be accepted as undeniably true that whenever X-rays or other electrical measures can cause more or less absorption of scar tissue, these same agents, properly employed at an early stage, would have minimized its formation. Such treatment involves the use of bedside apparatus, and, to conduct it on a large scale, it is necessary to have a special ward and practically duplicating the electrical department. Various difficulties of a financial and administrative nature prevented this bedside treatment of septic wounds from being given a full trial during the war. My own experience of it at Aldershot—where I directed the electrical service for a considerable time during the progress of hostilities—is limited for the most part to amputation stumps, and to face wounds in the plastic surgery wards at the Cambridge Hospital, where

Major Harold Gillies had his cases previous to his taking charge of the special hospital at Sidcup. The results obtained were, however, quite sufficient to show that the proper course to follow is to secure that the progress of a septic wound is normal day by day, rather than to seek to remove the end results of vicious healing. To accomplish this requires daily inspection of the wound, and close co-operation between the surgeon and the electrical expert. It is the business of the latter to prescribe and vary the electrical procedures, but he may from time to time call on his colleague for minor surgical measures—for example, the removal of a small slough, the curettage of a sclerosed portion of a wound or the re-opening of a too-rapidly closing sinus.

Plastic surgeons have certainly shown an excellent example in the matter of combined treatment. Not only have they sought the aid of the electrotherapeutist with regard to the bedside treatment of septic wounds, but also in cases of delayed union in simple fracture of the lower jaw. In the absence of sepsis, necrosis, mechanical obstacle, or malformation, non-union indicates a failure of the bone to respond normally to the stimulus of fracture. The bone is a living tissue whose functions are as readily modified, for good or evil, by local or constitutional causes as are those of any other tissue. Properly applied electrical stimuli always tend to jolt a pathological functioning back into a normal functioning, no matter in what direction the variation from the normal has oc-

curred. It is therefore not surprising that application of the high-frequency vacuum electrode over the site of a fracture, or the passing of a galvanic current in its neighbourhood should frequently bring about the desired result.

This subject of non-union is obviously of great importance to anyone who is either in general or surgical practice. It rarely happens that treatment by galvanism, at any rate, cannot be made available, and as it involves no risk it should always be given a trial.

From a consideration of fracture, one is naturally led to the question of adhesions. In most electrical departments much time is spent in attempting to relieve adhesions about joints. Here, again, would it not be better to try to prevent their formation? This is, of course, already attempted by massage and early passive movement, but no one would claim that these measures are wholly successful, especially where a joint is actually involved in the fracture. A preventative measure to which no surgical exception can possibly be taken is the exposure of the joint to X-rays while splints and bandages are *in situ*. If this be done, it will be found that, even in complicated fractures about the elbow, there is surprisingly little limitation of movement—apart, of course, from any due to actual displacement of fragments.

As to what I have said so far, I speak from personal experiences. But it is my firm belief that peritoneal adhesions—the bugbear of the abdominal surgeon—could be to a great extent pre-

vented by post-operative raying. Certain it is that symptoms of a kind usually held to be caused by adhesions can often be relieved by radio-therapy; and prevention is proverbially both easier and better than cure. The proposition that X-rays would tend to prevent the formation of abdominal adhesions is obviously a difficult one to prove. A comparatively small percentage of the patients operated on develop serious trouble of this nature, and it would be necessary to treat a large number of cases, and to observe them over a period of months, or even years, before definite conclusions could be arrived at. Nevertheless, the proof should be attempted, as the amount of radiation required could do no harm.

This is a suitable point to enquire what it is that the X-rays accomplish when suitably applied. There is a Greek fable about a traveller who sought shelter from a storm in a satyr's cave. His hands were stiff with cold, and the satyr, observing that he blew upon his fingers, asked him why he did so. "To warm my hands," the traveller replied. His host then hospitably busied himself in preparing a hot drink, which, as soon as made, he handed to him. It was, however, too hot to drink, and the traveller blew into it as he had done against his fingers. "Why do you blow again?" said the mystified satyr. "To cool the drink," replied his guest. Whereupon, crying out that he could be no honest man, the satyr in a fury fell upon the traveller and slew him. Now, the radio-therapist is much in the position of the traveller. He uses

X-rays to assist in the formation of fibrous tissue, as in cancer; to destroy it, as in keloid; and there are those who would like to fall upon him as no honest man. Yet the explanation of the seeming paradox is simple: in both cases the stimulus provided by the X-rays gives nature the necessary impetus towards healing in her own way.

In so far as nature herself, partially or wholly, destroys a malignant growth, she does so by strangling the cancer cells by connective tissue. But in the case of a keloid, the connective tissue has itself taken on a pathological form, and a return to normal requires its absorption. This tendency to restore balance, to soothe or stimulate, augment or repress, according to what is needed, applies to electrical treatment in general; except when a definite suppression or destruction is aimed at, in which case one is really dealing with a form of surgery. For instance, X-rays may be used over a considerable period of time to regulate ovarian function where there is excessive bleeding at the menstrual periods; or a few massive doses may be given which completely and permanently stop the ovarian function. In this latter case a kind of bloodless ovariotomy is in reality performed, which is not without special risks of its own, and the whole proceeding partakes of the nature of a surgical operation.

A simple analogy may help you to understand the essential difference between radio-therapy and what I may call radio-surgery. A plant which has been suffering from lack of light will benefit in all

its functions by exposure to a suitable amount of sun; but if we concentrate sunlight on a particular leaf or organ it will wither and die in a period long or short, according to the excess of light which is allowed to fall upon it.

I have gone into these theoretical questions at some length because some medical men still fear that actual harm may be done by X-rays if they are used for the various purposes for which I have advocated their employment. It is, of course, true that grave injury may result from faulty methods; but the same may be said of any powerful therapeutic agent.

Accompanying the use of radio-surgery—using the term in the sense I have defined—are certain risks which must be duly weighed against the looked-for results—results as definite as those obtainable by the knife. I use the word "results" rather than "benefits" of deliberate purpose. The surgeon may remove a man's appendix: the disappearance of the organ is a definite result from the operation. But the patient is not necessarily cured of his complaint. Similarly, the permanent destruction of ovarian function can always be accomplished in women over 40—this is the result of radio-surgery; but if disturbed ovarian function is not the cause of the patient's symptoms, or only in part the cause, again cure will not result. The risks of radio-surgery are: injury to the skin, usually trifling, but sometimes serious; and possible injury to other organs besides those which it is desired to affect. But where the in-

dividual doses are small, and the course limited to a few weeks, the patient runs no risk in the hands of a physician. If no good is done, at least the patient has had the chance of benefit, and is no worse than previously. This is so even in cancer—except in certain advanced cases where risks are justifiable. The pre-operative treatment previously advocated does not in any way interfere with subsequent normal healing.

The agents included under the general term "electrical" are X-rays, light, high-frequency, and static discharges, galvanism, faradism, and various modifications thereof. The use of X-rays and light in conjunction with surgery I have discussed at some length, and I propose to devote the remainder of my time to a consideration of the combined treatment of infantile paralysis, into which treatment X-rays do not enter.

American writers claim that the high candle power lamp applied over the spine is of use in the acute phase of the disease: of this I have no personal experience. As to the more chronic stage, the mild case comes almost wholly into the domain of physical medicine; the very severe case is largely a surgical problem, but there are few patients who cannot derive some benefit from physical measures. Leaving aside the few instances in which a late relapse is believed to have occurred, we are dealing in this disease with a destructive process which ceases after a few hours, or, at most, a few days. The extent of the initial paralysis is well known to be no guide to progno-

sis, except in the sense that the worst possible is known, but not the best. A very common result is for a lower limb to be still more or less seriously paralysed when the period of natural recovery seems to be coming to the end.

The treatment of a case not requiring surgical interference must first be briefly dealt with in order that the *rationale* of combined measures may be understood.

First, deformities must be prevented by suitable appliances; secondly, all muscles which can be made to contract by any form of electrical stimulus must be systematically exercised, and, thirdly, the limb as a whole should be stimulated by high-frequency applications, both locally and to the spine. If all the cells in the affected region of the cord were either dead or capable of spontaneous recovery, electrical treatment would be useless. But there are, as a matter of experience, always numerous cells which remain, as it were, in a state of suspended animation; and it is these which the electrical stimulus can call to life. Unfortunately, they often tend to become sluggish after a few months, and the electrical excitation must be continued at intervals until adult full growth is attained.

There is sometimes a tendency to be concerned too much with the mechanical side of a case. Deformities are corrected, tendons are transplanted from muscles more or less healthy to do the work of those which are paralysed, and apparatus is prescribed to give mechanical support when neces-

sary. But when all is done the limb too often remains blue, cold and shrunken, the transplanted tendons insufficiently energized properly to carry out their new work, and year by year the difference in size between the sound and the affected member becomes increased rather than lessened.

Massage and exercise are always prescribed, it is true, and the days are gone by when the value of electricity in the treatment of paretic muscles can be questioned. The electrical stimulation of paralysed muscles has, in fact, become orthodox, and already it has been almost forgotten that electro-therapeutics only a few years ago were fighting tooth and nail for its acceptance. The doctrine of the stimulating effect of electricity on the growth of limbs is not yet canonical, but it is neverthless true. With the exception of a few rare cases in which only a single important muscle is affected, it may be said that any limb sufficiently injured by poliomyelitis to need surgical attention is also in need of electrical stimulation. A six weeks' course will often result in a spurt of growth lasting several months; and if the stimulus be repeated at suitable intervals over a period of years the development of the injured limb may lag but little behind that of its fellow.

Combined treatment must be carefully distinguished from blunderbuss therapy. This latter may be defined as the simultaneous employment of a whole host of drugs or methods, each individually of doubtful utility, in the hope that, in the mass, they may accomplish some useful purpose.

In combined treatment we employ agencies each of which has been separately tested and found to be of definite value, often to the point of actual cure, in the malady to be attacked.

And we make use of these various methods in such a manner and in such sequence that they amplify and reinforce each other.

Let us remember that Nature recognizes neither the surgeon nor the radiologist, nor the expert in drugs, nor yet the lord of germs. She will put forth her best efforts only when she is assisted without stint. General practice is in reality the only logical form of practice. But no human mind can compass the whole field of medicine. Hence the hope of the future lies in specialism tempered by co-operation.

ON THE AFTER-EFFECTS (AND SO-CALLED AFTER-EFFECTS) OF ANÆSTHETICS.

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Some years ago a surgeon said to me: "Cannot you anæsthetists do something to avoid this horrible after-vomiting?" This led me to pay particular attention to after-effects frequently attributed to the anæsthetic, with a result which was unexpected, for I found that so to attribute them, at any rate entirely, is frequently to make a considerable mistake.

Such complications are, like fatalities occurring during anæsthesia, due to the anæsthetic in variable degrees. In some cases the anæsthetic, or its maladministration, may be the sole cause; in others, it may have contributed to some extent; in others, it may be hard to trace to it even a remote share in the responsibility.

Before dealing with after-effects, I will say a little about the intermediate, or recovery stage, for at this period so-called accidents and even fatalities due to mistakes or inattention are more common than they should be.

A faulty position—for example, the arm twisted under the body, may result in paralysis of nerves, or there may be severe burns by a hot-water bottle. The patient may close the lips and clench the teeth, and if the nose is blocked be unable to breathe; or he may choke in vomiting—or if left alone may try to sit up and fall in a faint, or may bury his face in the pillow and be suffocated. When possible the head and shoulders should be turned well to the right side and slightly raised, so that the tongue does not fall back and fluid is more easily expelled. If there is likely to be trickling of blood from the nose and throat, this, or a semi-prone position, is essential to safety; if lying flat on the back one does not know how much blood is being lost; it may form a clot, blocking the air passages or collect in dangerous quantity in the stomach.

If semi-conscious, dressings may be displaced, or the wound infected or torn open.

A patient should therefore never be left, even for a moment, until fully conscious, and the nurse should be instructed in her duties in case of emergency. If necessary the lips should be separated and the uppermost cheek pulled away from the teeth, allowing air to enter behind them; a wedge or gag may be needed. The tongue should *not* be drawn out nor the jaw pushed forward during vomiting, thus opening the larynx, unless there are signs of asphyxia, in which case the throat must be swabbed out at once.

It has been urged as one of the advantages of

prolonged administration of nitrous-oxide and oxygen for major operations that the patient recovers consciousness far more quickly than after any other anæsthetic. A speedy return to consciousness, however, is not always desirable, even after minor operations, particularly when pain may be expected to last for some time.

As humorously remarked by an experienced anæsthetist at a discussion: "We have been told that the patient can talk to the surgeon directly the operation is finished, but we have not been told what he says to him!" In fact, too early consciousness of pain, especially after abdominal operations, and when there is tension, has been stated by some surgeons to be a considerable objection to the use of this method; and it cannot always be obviated by the use of local analgesics or morphine.

Depression and collapse should not be directly traceable to the anæsthetic save in quite exceptional cases. As a general rule, the previous state of the patient, hæmorrhage, stridor or other reflex disturbances leading to surgical shock, are quite enough to account for the condition, and remedial measures should be under the direction of the operator. If, however, the anæsthetic has been improperly selected or badly given, its administrator must be considered responsible: say, if chloroform (to give an extreme instance, but one that actually occurred) is given by irregular dropping to an anæmic girl for extraction of several teeth.

Excitement and struggling is less usual on

recovery than during induction, but may occur in neurotic or alcoholic patients, especially if they are disturbed by noises or otherwise. An attack of *delirium tremens* may be precipitated; but here the direct effect of the anæsthetic counts for little in the causation.

Insanity rarely follows immediately, but may come on during the first two or three weeks after operation in predisposed subjects, or after operations on the brain or generative organs, when the anæsthetic may perhaps be considered to be one cause among many others.

If a person has formerly been insane, only imperative operations should be done; but in some chronic cases there is no more liability to ill-effects than in sane people, and less emotional reaction. Other cerebral disorders, such as localized softening, are unusual. They may be due to vascular disturbance or perhaps to the toxic effect of the anæsthetic.

Vomiting.—This in any marked degree causes much discomfort, and may spoil the work of the surgeon, or even lead to death by exhaustion or visceral extravasation. Predisposing causes are numerous. It is more apt to occur in sensitive people, in dyspeptics, in the over-fed, and in those whose excretions are inadequate. Faulty preparation, oral sepsis, swallowing of mucus, or blood, also faulty after-treatment, such as jolting, feeding too soon, etc., often contribute. The disorder for which the operation has been needed, such as

intestinal obstruction, may not have been fully relieved.

The surgeon's proceedings may unavoidably cause after-vomiting, by direct or reflex action, and operations and bandages may interfere with oxidation and elimination of the anæsthetic and of waste products. If sensitive organs are stitched, it is more apt to occur than after their removal, and it may be associated with spasmodic pain—for example, passage of clots down a ureter. One must beware of after-complications, of which vomiting is a symptom, such as peritonitis, or intracranial mischief. Any operation in the region of the diaphragm inhibits its action, thus allowing atonic distension and nausea.

Prolongation of the anæsthesia I do not find much increases the tendency *per se*. It must be remembered that in case of a long operation other causes for after-vomiting more usually exist than in that of a short one.

Now, I wish you to understand that, contrary to the prevailing impression, sickness is *not* an invariable result of taking an anæsthetic, and should not occur in any marked degree, unless there have been other unavoidable causes, such as I have indicated.

In a series of over 1,000 cases of my own, from which I excluded short anæsthesias, I found that there was no after-sickness in three-fifths, including many long and severe operations, and it was inconsiderable in the majority of the rest; in only a small percentage was it marked, and in them was

fully accounted for by peculiarities of the patient, or the nature of the operation.—(*Lancet,* 1911 and 1913).

I am, at the same time, far from denying that an anæsthetic, improperly selected or improperly given, may be the direct cause of severe vomiting.

Nitrous-oxide or ethyl-chloride is not, as a rule, followed by vomiting, unless blood is swallowed. After ether, vomiting is more usual, but often only once, and before return to consciousness. There is no special tendency to vomiting from C.E. mixture. Chloroform is less often followed by vomiting than ether, but when it does occur it is more apt to be prolonged and serious. There is a condition known as "delayed chloroform poisoning," of which vomiting is usually a marked symptom. It occurs chiefly in children. After an interval of some hours, during which the child is apathetic, persistent vomiting starts, with delirium tending to coma and death. There is generally acetone in the urine, and a characteristic odour of the breath. The pathology is still unsettled, but it seems that carbohydrate starvation, and overwork of the liver and kidneys in eliminating toxins predispose, particularly when there is acute sepsis, previous malnutrition, and depression from hæmorrhage or inability to take nourishment after operation.

If retching and vomiting recur, a small tumblerful of hot water, in which a teaspoonful of bicarbonate of soda is dissolved, should be slowly sipped. This will clear the stomach one way or the other. The mouth should be rinsed, also a

thin slice of lemon sucked and aromatic vinegar inhaled if there is complaint of the taste and smell of the anæsthetic.

I have not much faith in remedies such as black coffee, iced champagne, mustard to the epigastrium, of which a long list is commonly given. They may have some effect in emotional patients, but are useless in serious cases. If danger threatens, one should not, as seems to be the usual practice, try one after another a variety of sedative drugs and domestic remedies, but consider what may be the cause or causes, and take steps accordingly. It is seldom that the anæsthetist is called into consultation, as he should be in such difficulties. The urine should be tested, especially if there is possibility of acetonuria or uræmia. Examination of the fundus oculi may give early indication of intra-cranial disorder. Presuming there is no evidence of any condition which may suggest further operation, sedatives, such as bromides, may be given to neurotic patients, or morphine if there is persistent pain and irritation, but this drug must be used with caution, for if sickness depends on imperfect elimination, it makes matters worse, and it is also liable to cause intestinal atony and mask complications.

Inhalations of oxygen will do good if aëration is for any reason inadequate.

Lavage of the stomach is especially valuable when there are likely to be decomposing contents, and I would urge that it should be done as a matter of routine after (if not before) any operation

for intestinal obstruction. The bowels should be well cleared. If there is atonic distension, strychnine, ergotinine, eserine, pituitrin are recommended, unless the patient has high blood pressure or a weak heart; also abdominal massage when possible; and a rectal tube may be left in for an hour or so at a time. The "Fowler" or sitting-up position is advisable.

Glucose not only obviates and combats acid-intoxication, but is a food which makes no demand on the digestion. If necessary it may be given per rectum or intravenously (2·45 per cent.), and as a prophylactic, especially in case of children.

Pulmonary Complications.—It was shown by Dr. William Pasteur, in 1911, in his oration on "Post-Operative" (*N.B.*, not "Post-anæsthetic") "Lung Complications," that these are mainly due, not as was formerly supposed to the anæsthetic, particularly to the irritating vapour of ether, but to inhibition of the action of the diaphragm consequent on operations in its region, and tending to collapse of the lungs and broncho-pneumonia. Careful examination of the chest before and after such operations is therefore very important.

There are many other predisposing and exciting causes, such as catarrhal and septic conditions of the air-way, lack of general resistive power, chill, over-dosing with ether, inhalation of fluid, the use of dirty apparatus; the dorsal position, and hypnotic drugs. Œdema of the lungs may follow excessive intravenous infusion, and pulmonary embolism, septic or non-septic, may occur, especially after operations involving large veins.

Careful selection and administration of the anæsthetic, measures directed to secure a free airway and prevent inhalation of fluids, and the use of oxygen and atropine in certain cases, are obvious safeguards.

I have tried to show that ill effects following an operation under anæsthesia are by no means due to the anæsthetic to such an extent as is still commonly supposed. This mistaken impression is regrettable, for sometimes an anæsthetist is blamed when he should be excused, sometimes excused when he should be blamed. Moreover, vomiting and so forth being regarded as more or less inevitable, treatment is apt to be perfunctory and ill-directed.

By systematic inquiry and better co-operation it may be hoped that such evils will be more often avoided, or, if they must happen, more effectually remedied. The responsibility of the anæsthetist will thus be increased rather than diminished, because it will be more strictly defined.

CHRONIC PAROXYSMAL TRIGEMINAL NEURALGIA AND ITS TREATMENT.

(A Clinical Lecture.)

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The subject of these lectures has been revised and brought up to date in order to present results of my experience of the treatment of this disease during the last twelve years.

Trifacial neuralgia, chronic trigeminal neuralgia, tic douloureux, or neuralgia major of the fifth nerve, as it has been variously described, is an inveterate and violently painful paroxysmal neuralgia, affecting, in the majority of sufferers, one side of the face only. In by far the larger number of sufferers the pain is referred to the territory of the second or the third division of the fifth nerve—that is to say, roughly, the upper jaw and cheek, upper lip and side of the nose for the second division, and the lower jaw, lower lip, and perhaps the side of the tongue for the third division. In a considerable proportion of cases the second and third divisions of the nerve are both affected, and the pain will not cease, or ceases only for a time, if one

of the divisions alone is treated. In a small proportion of cases the supraorbital branch of the first division is involved, scarcely ever alone, but in addition to neuralgia affecting the second division or the second and third divisions. Supraorbital neuralgia alone is usually due to temporarily acting causes, such as influenza, malaria, or other fevers, or to such causes as errors of refraction, frontal sinusitis, migrainous neuralgia, and many others, and it must be distinguished from the chronic paroxysmal and inveterate disease known as trigeminal neuralgia. The pathology of this disease is somewhat obscure. What is certain is that there is no disease to be detected by hitherto known methods, either in the nerve centres of the brain, Gasserian ganglion, or main branches of the fifth nerve. The undoubted fact that efficient interruption of the conduction of afferent sensations along these branches of the fifth nerve gives instant relief to the neuralgia appears to me sufficient proof that such neuralgia is dependent upon stimuli affecting the nerve endings at their periphery.

The results of destruction of the ganglion as compared with neurectomies or alcohol injections below the ganglion differ only in the degree of their permanence—that is to say, operations on the nerve trunks, however complete at the time, are always followed by some re-establishment, sooner or later, of nerve conduction through downgrowth of nerve fibrils from the ganglion cells above. If, on the other hand, the ganglion is thoroughly destroyed by any means, the anæsthesia of the fifth

nerve territory is permanent, and the cure of the neuralgia is correspondingly complete.

The nature of these stimuli from the periphery in this neuralgia is a matter of speculation, but it seems highly probable that they are due to septic neuritis of dental nerve filaments, the result of previous dental caries, pyorrhœa, or abscess around the roots; and recently in a case of this chronic neuralgia a piece of the maxilla was removed for me by Mr. Warwick James under the most careful aseptic precautions, and a pure growth of streptococci was obtained.

I have been impressed, in taking the histories of cases, by the number in which the pain has definitely started immediately after dental operations or antral abscess. No other nerve in the body, except the second and third divisions of the fifth nerve, is so liable to chronic infection of its branches, and although in some cases even many years have elapsed between the loss of a tooth and the onset of the neuralgia, yet this does not exclude septic neuritis from dental infection as the cause, and when the infection has thus spread backwards into the dental filaments within the jaw it is easily understandable that removal of the tooth does not cure the neuralgia; indeed, it often makes it worse.

Causes.

During the last twelve years I have seen a large number of cases of chronic trigeminal neuralgia, and I have analysed my records of over 312 cases, 230 in private practice and 82 hospital cases. I

have, in addition, seen and operated upon a considerable number of hospital cases whose records are not available or are inadequate. Some curious points come out in my analysis which I have never seen referred to in other writings on the subject. The proportion of women affected to men is 62 per cent. to 38 per cent., or about three women to two men, while of the unilateral cases of neuralgia, which of course form a very large majority, the right side is affected in correspondingly greater frequency—namely, 62 per cent. to 38 per cent. This excess of right-sided cases over left-sided obtains in each group of men and women, both in private cases and in hospital cases, and moreover, in each separate year of the eleven years I have analysed a somewhat excess of right-sided cases over left is seen. I cannot therefore look upon this difference in frequency of incidence on the two sides as accidental; what its cause can be it is not easy to say.

I do not know whether any statistics are available of the relative frequency of dental caries on the two sides. Other factors, too, have to be taken into account which may act as the exciting cause for the origin of trigeminal neuralgia. I have already spoken of dental operations originating the pain in a certain number of cases, such as fracture of teeth and difficult stump extractions. In ten of my cases severe *chill* to the face, as by motoring in a cold wind, was the immediate precursor of the original spasms of pain; in five cases intense *emotion* immediately preceded the first attack. A

woman was shocked by being told that her child, who was playing in the roadway, had been run over by a motor car and taken to the hospital. She immediately hurried off to the hospital, and on reaching there three-quarters of an hour later heard that her child was dead; at the same time a spasm of violent neuralgia struck her in the right lower jaw, the pain continuing at intervals until I saw her two years later, and was able to arrest it by injecting the third division of the fifth nerve with alcohol.

Still another cause of onset of this terrible neuralgia is a *blow* upon the jaw or face; this occurred in six of my cases. For example, a young woman, aged 27, was stooping to pick something off the floor when another girl's head struck her violently on the lower jaw, making her teeth rattle, as she said. About three hours later, when she had just mounted on to the top of an omnibus, she was suddenly seized with the most excruciating pain in the face, which made her behave so wildly that she said she was like a wild woman, and had to be taken off the bus. The paroxysm lasted altogether some ten minutes, but recurred again and again in the manner characteristic of trigeminal neuralgia until I saw her and injected the third division of the nerve some years later.

Such suddenness and violence of onset of the spasms of pain are extremely characteristic of this peculiar form of neuralgia, and I have notes of some forty-six cases in which this was the case. One of apparently the most severe cases of pain I have ever seen began in this way. She was a

cook, aged 45, who in her spasms of neuralgia lost all control, and screamed and threw herself about. Six years previously her first attack occurred, without any warning one night at 3 a.m., when she was awakened with the most awful agony in her face. Her screams awoke the whole household; a doctor had to be sent for, and she was kept under morphine almost continually for three weeks. She got no relief from any drugs until I injected the second and third divisions of the fifth nerve with alcohol; this gave her complete relief for two years. Recurrence and reinjection with alcohol again gave her two years' freedom from pain, when I injected her Gasserian ganglion with alcohol, and since then she has been completely free for several years.

In other patients the pain may at first be mistaken for dental neuralgia, but the spasms of pain gradually increase in severity, in spite of the removal of teeth, until at last it is realized that the neuralgia is of the graver inveterate type.

Age.

The age of the onset of the disease varies very widely in my experience, the oldest being 85 when the disease commenced. In two cases, both females, the severe neuralgia began at the age of 17; one of them had suffered from neuralgia of the third division for twenty years when I saw her, temporary relief only having been obtained by a division of the inferior dental nerve. Alcohol injection of the third division ten and a half years ago gave her complete relief, which still persists. The

other patient was a lady aged 70 when I saw her for the pain; she had suffered almost constantly for fifty-three years; in her case injection of the ganglion was done under chloroform two and a half years ago, causing total anæsthesia of the fifth nerve and complete relief of all pain, which should be permanent.

The onset of the neuraligia is rare under the age of 30, though I have seen eleven other cases commence before the age of 30, at the ages of 20, 21, 22, etc. The average age of onset in 205 of my cases is almost exactly 50. Once the disease has started it is practically certain that permanent cure can only be obtained by destruction of the nerve trunks involved, or of the ganglion itself. In only two instances have I known the neuralgia disappear spontaneously, after existing a number of years. One was the father of a lady whom I injected for typical neuralgia, and she told me that her father suffered from the same spasmodic pains from the age of 65 until he was 90, when the pains gradually left him, so that he was free from the neuralgia during the last twelve years of his life, for he died at the age of 103.

In a number of cases a considerable interval may elapse between the first onset of the neuralgia and its subsequent establishment. A lady had severe spasmodic pain in her left lower jaw at the age of 45, lasting for nearly two years, when it left her for thirty years, recurring severely after exposure to a cold wind when driving. After the pain had continued for nine weeks I injected the left Gasserian

ganglion. I have notes of similar intervals of twenty years, seventeen years, nine years, seven years, and so on.

Heredity.

I have noticed heredity as a factor of trigeminal neuralgia in five cases. I have already referred to one patient, a lady of over 60, whose father suffered for over twenty-five years from this form of neuralgia, and then outlived it. In another instance I have treated a mother over 70 years of age, who suffered for many years with bilateral neuralgia, mostly of the third division. Injection of the nerve at the foramen ovale gives her about two years' freedom from pain. One of her daughters at the age of 36 began to suffer in a similar manner with spasmodic neuralgia of the right third division. I saw her for this soon after the pain began, and recommended drug treatment and rest; this, however, failed entirely. I did not see her for three years, the pain having continued all that time, and she told me she had been twice injected by another physician without any relief, as the nerve could not be found. I then injected the third division at the foramen ovale, causing complete anæsthesia of its distribution and immediate cessation of the pain, from which she has now been quite free for three and a half years. In my other three patients—two men and one woman—it was the mother in each case who was said to have suffered from typical trigeminal neuralgia for many years. In investigating such histories one must be careful to dis-

tinguish between migraine and the true trigeminal neuralgia.

Chill.

I have already referred to chill as being the exciting cause of the first spasms of the neuralgia in ten cases. One illustration will suffice. A woman, aged 50, drove twenty-five miles in an open dogcart through a blizzard, snow, and icy cold wind to Exeter. The same evening, about 9 o'clock, while she was having supper, intense neuralgia struck her lower jaw, the pain being on the same side as had been exposed to the wind. This was the bebeginning of a severe case of trigeminal neuralgia which continued for several years until I saw her. In numerous other instances exposure to a cold wind or draught will restart the paroxysms of a neuralgia which has become dormant, and, indeed, cold wind or draught may be said to be the worst enemy to these subjects of neuralgia.

Although chill has such a bad effect upon trigeminal neuralgia it is surprising that subjects of this disease have not special immunity during hot weather, and it is extremely common for patients to say that the time of year makes no difference to their pain, and it is waste of time and money to send such patients for treatment to a hot climate.

"Warnings."

I have referred to the absolutely sudden onset of the disease in numerous cases. Three of my cases have developed a warning by which they recognize

the imminence of a period of severe attacks. A lady, aged 64, had suffered from right lower jaw neuralgia for ten years, made worse by extraction of the teeth. Alcohol injections by Horsley gave no relief. As she says no numbness of the face was produced, the nerves were no doubt not injected. She says she has always noticed a burning sensation on the top of the head for some days before the neuralgia attacks commence. Alcohol injection, in May, 1916, of the second and third divisions relieved her pain entirely, the relief still persisting. In another case, a man whom I injected at the age of 39, with neuralgia of the left second and third divisions, seven years ago, used to get a warning, for a day or two before the neuralgia attack, of constipation and sensation of swelling in the gums.

Another patient, a lady aged 54, says she always gets a shivering sensation along the right cheek before the pain starts. She had lost all her teeth twenty-seven years before the pain began.

Character of the Pain.

The suddenness of the onset of neuralgia is often likened to an electric shock; one patient thought she had bitten on to a needle, another that she had broken a tooth. Some speak of a stinging pain at the commencement. Very many describe the pain as hot, "like a lot of red-hot bradawls being pushed up," "flickering," "hot," "like daggers," "like a hot knife or needles being run into the forehead." In others the pain starts with a tacking sensation

or tapping, scarcely painful at first, but rapidly getting worse, until the pain "explodes along the nerve like fireworks or hot electric wires." The severity of the attacks must be witnessed to be appreciated, and patients get little sympathy or credit for their suffering, except from others who have seen them in it. Some patients lose all control of themselves in the attacks, screaming out, fighting, and struggling. In others the jaw becomes fixed, perhaps wide open, and saliva may trickle from the mouth.

One patient described the commencement of her attack as having suffered for two days with severe agony in a tooth; she thought such pain was impossible, had two teeth out, but was no better for a week; since then she had been subject to repeated attacks in which the pain stings into the right side of her tongue. She calls it a "hellish pain," and says it has destroyed her nerve, and she feels during the attacks as if she could smash everything, or commit suicide. Indeed, before the remedy was devised for this pain in the operation of gasserectomy, and later by alcohol injection of the nerve trunks, suicide was not a very uncommon ending for sufferers from this dreadful disease.

So long as patients are able to take food they do not lose weight or suffer notably in health, but as soon as the neuralgic spasms are sufficiently frequent and severe to prevent the taking of food there is danger of rapid loss of strength from starvation, and it may be necessary for the patient to suck liquid nourishment through a straw or

tube on the other side of the mouth, and to write instead of speak, because the spasms of pain are started by attempts at eating and talking.

Treatment.

Only a comparatively small number of sufferers from trigeminal neuralgia get relief from drugs. In a few gelsemium gives relief, though with the majority even morphine in large doses fails. Practically the only two methods are gasserectomy and alcohol injection. In my opinion the former operation should never be undertaken until alcohol injection has been thoroughly tried. During the last ten years I have injected the Gasserian ganglion with alcohol through the foramen ovale in sixty-three cases; in thirty-one the anæsthesia has remained total and no recurrence of pain has taken place. In many of the remainder partial ganglion anæsthesia has persisted though pressure could be felt, yet the relief from pain appears to be equally good.

In many cases, though total ganglion anæsthesia is produced at the time of injection, yet the anæsthesia of the first and second division wears off, perhaps completely, in from ten minutes to an hour, though the third division remains totally anæsthetic. This is no doubt due to the ganglion cells having been shocked, and not destroyed, by a partial infiltration only. In such a case pain is liable to recur after twelve months or two years, as in a case of ordinary injection of the nerve. With total destruction of the ganglion there is the

same liability to keratitis as with the operation of gasserectomy, but if the lids are kept carefully closed by strapping for the first week and the conjunctival sac washed out with boracic lotion twice a day, I have found little or no trouble with my later cases. The lids, however, should be kept closed from the beginning, and it is unwise to wait and see whether keratitis develops, as then it will be necessary to keep them closed for a much longer time.

Hutchinson's partial gasserectomy avoids the danger of keratitis, but is not a certain permanent cure, as I have seen three such cases relapse with neuralgia.

Bilateral Neuralgia.

Bilateral trigeminal neuralgia is comparatively rare, though I have seen and injected twenty-five bilateral cases, usually at different dates, but on several occasions I have injected both sides, one after the other.

Gasserectomy is an impossible operation for bilateral cases of trigeminal neuralgia, owing to the necessary destruction of the motor branch of the fifth nerve on each side and consequent jaw-drop. Alcohol injection has the advantage that the nerve injury is less complete, and the motor nerve fibres regenerate. In one case I produced jaw-drop for two and a half months, which recovered completely later. On December 10, 1919, I injected the left Gasserian ganglion in a woman of 49, for neuralgia of the left upper jaw, whose

right Gasserian ganglion I had previously destroyed by alcohol injection seven years previously. She was still totally anæsthetic over the whole right fifth distribution, just as much as in any case of gasserectomy, though in her case the motor branch had recovered well, and she had quite good right temporal, masseter, and pterygoid muscles. Injection of the left Gasserian ganglion has now produced in her case total anæsthesia of the whole of the face and front half of her head, though she can chew fairly well by means of the muscles on the right side, and those on the left will doubtless recover also later. So far as I know, bilateral destruction of the Gasserian ganglia has never previously been done.

Method of Injecting Alcohol.

As regards the methods of practising alcohol injection, details have already been published and need not here be repeated, though I would like to insist on the advisability of using the method of trial and test. That is to say, when the needle is thought to have struck the nerve inject a few drops of 2 per cent. novocain, and, after waiting half a minute, test the skin distribution of the nerve for anæsthesia. If none has developed do not inject alcohol, but try again to find the nerve. As soon as anæsthesia appears to novocain injection, then complete by injecting about 1 c.cm. of 90 per cent. alcohol, when total anæsthesia to touch, pin, and pressure will develop almost instantaneously if the

nerve at the foramen ovale or foramen rotundum has been correctly hit.

Results.

In almost every case, as soon as anæsthesia develops from the injection, the neuralgic spasms cease, and they can no longer be started by any chewing movements or rubbing the face.

The duration of the cure rarely lasts less than twelve months, if good anæsthesia has been obtained, and in the majority the relief from pain lasts from two to three years. Very many of my cases have been quite free for four or five years, one six and a half years, another seven years, another nine years after nerve injection only, while I expect my cases of ganglion injection to be permanent cures, just as if gasserectomy had been done. No trophic effects on the skin are ever seen. Keratitis is a risk if the cornea is anæsthetic after injection of the ganglion, but this may always be avoided by proper precautions.

MORBID MENTAL GROWTHS.

By Sir GEORGE H. SAVAGE, M.D., F.R.C.P.[1]

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The subject I have chosen for my lecture is one that has interested me for very many years. Quite early in my experience at Bethlem Hospital I was struck with the frequency with which I was able to trace a connection between some definite delusion and some real experience in the life of the patient. Later, I began to notice that there was, not infrequently, a relationship between the mental disorder and some dream condition. Lately, of course, this has been very much more strongly brought to our notice by the researches of Freud and others on psycho-analysis. Early on, as I say, I was impressed by the fact that a dream might start a mental disorder, which it coloured throughout. Perhaps the most striking example I remember was that of a governess who was in Bethlem Hospital, a very refined and educated woman who wrote a very full account of the development of her disorder. According to her, she had lived innocent of sexual conditions until about 14 or 15 years of age, when she had an erotic dream; and from that time onwards erotic dreams recurred, and finally established a hysterical and

[1] Deceased since the delivery of this lecture.

emotional eroto-mania that rendered it necessary for her to be sent to an asylum.

I have seen similar delusions arise in women at the menopause: sexual dreams with such patients have led to the idea that they have been indecently assaulted, that they have been, in fact, drugged and then ravished. A fair number of such cases have to be put under control because, not only do they develop the idea, but they may make definite accusations against some innocent person. In more than one such case the unfortunate person has been a doctor.

Again, I recall the case of an elderly lady who, as not infrequently occurs with senile mental changes, re-developed a considerable amount of erotic feeling. She was a very religious woman, and the idea that she should have these lustful and evil thoughts greatly distressed her, until one night, after a very marked erotic dream, she was convinced that a personal devil had had connection with her, and that she was, in fact, pregnant by the Devil.

As I have already said, the occurrence of mental disorder associated with, if not dependent upon, dreams leads one to the consideration of the psychic analysis of Freud. I shall, more fully later, trace some relationship between the two, more particularly in reference to the treatment of these cases.

When considering the subject of morbid mental growths, I have made three classes: first the simple morbid mental growth, affecting really al-

most solely the individual; next the involving or invading growth, affecting not only the individual but affecting him to such an extent that society has to be protected from him. Yet in these cases there may be little, if any, intellectual loss; third, I shall refer to the malignant or destructive morbid mental growths, those, in fact, in which not only has some dominant delusion appeared, but in which the delusion has been associated with steady and progressive mental deterioration.

First, then, as to simple morbid growths. In some cases these morbid mental growths may be traced to a habit: what might appear to be quite a simple eccentricity may develop so far as to render the person not only of unsound mind, but actually insane. I have compared such conditions to the large fatty growths that are to be seen in surgical wards, growths that interfere with the patient getting about and yet do not seriously affect his general health. The habit may develop from some simple sensory defect. Thus, it has been pointed out that certain deaf people gradually become persuaded that, as a result of their deafness, persons gradually shun them or avoid them. This may lead to habitual isolation, and I have come across a fair number of patients who, with some slight personal defect, have become so persuaded that they were aliens that they have, for years together, secluded themselves. I remember one man of considerable intellect and a great art critic who, for twenty or thirty years secluded himself in his mansion, only going out at night. Yet,

in his case, there was no further intellectual defect. A simple idea may grow into a morbid delusion, and I have elsewhere described cases such as the following:—

An English governess in a Russian family recognizes that she, intellectually, is much the superior of the immensely rich Russian noble. Dwelling upon this, and being isolated because, from her position, she could not associate with the servants and was ignored except as far as her position as governess was concerned, she dwelt upon the inequality of things and what I may call the "might-have-beens" or "ought-to-have-beens," that if mind really was to be the ruler, *she* ought to be the princess and the other people ought to be subservient to her. Slowly this developed into the idea that she was actually a princess.

In another case I remember seeing a woman who was the wife of a gamekeeper to a nobleman. She, recognizing that she was better looking than the duchess, and that her education was quite a good one, felt it was altogether wrong that she should be in such a subservient position as that of gamekeeper's wife: and slowly there grew the idea that she was a person of noble birth.

Besides such cases, one has to recognize a more serious group, in which the self-feeling grows into marked hypochondriasis and I have seen a certain number of persons who have been intellectually bright and yet who have developed the idea of physical inefficiency. It is well known

that a great lady philanthropist spent many years of her life bedridden as a confirmed neurotic invalid. There was no doubt that she fully believed that she was incapable of getting about and of exercising her bodily strength. Yet, periodically and spasmodically, she would do the most arduous physical work and then collapse. I have come across a fair number of similar cases in which what may be called hysteria or may be looked upon as hypochondriasis, has ruined the life, although it has in no way affected the intellect, of the patient. Of course it is comparatively rare in medicine to find one, and only one, efficient cause, and one constantly meets with cases that seem to pass from the one class—the simple morbid mental growths—into that of the invading class. I might give, as an example, the following:

A man, who had distinguished himself both at public school and university and also had obtained a good position in the Civil Service, while living in a tropical and somewhat isolated place, developed grandiose ideas, and soon repudiated his social and family relationships, considering himself really to be of Royal or Imperial birth, denying his wife and repudiating his name. Yet, notwithstanding this and having to be sent to an asylum, he developed all sorts of new abilities: he was a recognized naturalist, and arranged the museum of the asylum. He also scoured the whole country for botanical specimens. Later, he devoted himself to the study of Oriental languages, and acquired great success in these studies. Yet

nothing could persuade this man to acknowledge his family or to resume any social contact with the world outside.

Here, then, we have examples of individuals who, through their isolation, sometimes from morbid sensations, have developed a series of delusions that have dominated or controlled their lives. Some of these individuals have never required to be placed under control; others have, for their own sakes, had to be certified. "If a man will not work, neither can he eat," and many of these individuals had simply to be secluded because they would not earn a living.

I now pass on to the consideration of invading morbid mental growths. Probably these produce some of the most interesting cases, cases in which there can be no doubt of the necessity of the individuals being under care and control. Yet for many years the patients may show no sign whatever of any further intellectual decay. Under this class I would place many of the obsessions. Psychic analysis has traced the origin of a certain number of these obsessions to subconscious or dream states. I have not had the opportunity of doing that, yet it is quite easy to understand how a simple fright and shock may lead to a very definite obsession. One case I saw with so-called "bladder stammer," who traced his trouble to a time when, as a small and rather weak boy at a public school, he said the bigger boys used to make fun of him when he went to the urinal, that they used to push him away. In that way

there was established a dread of going to the urinal, and this gradually grew into a complete obsession, so that he could not make water with anyone near him.

In another case—quite a natural thing—a man was in a comparatively slight railway accident, but the accident made such a definite impression on his mind that he was never able to throw it off; and although he could ride in a motor-car or in a "taxi," any travelling in a train would be always followed by vomiting, and even collapse. In an earlier stage of his disorder he, not infrequently, made efforts to go in trains, but they were always associated with the most abject terror and mental collapse.

In these cases there is a very definite starting point for a morbid mental growth. The thousand-and-one dreads and fears which have come before my notice—more particularly during the war—illustrate how readily a fact may grow into a delusion. Among these dreads I would take, as an example, the case of a man who, when he was in France, was bitten by a dog. At that time there was a rumoured epidemic of rabies. He was a wealthy man, and, in order to avoid all risk, he went into the Pasteur Institute, where he was completely treated. He came back, but the dread of dogs and of hydrophobia was such that whenever be became upset, as when he had a slight attack of influenza or had any physical malady, it always showed itself in a recurrence of his dread of rabies. His attention was, for a time,

diverted by getting him to take an interest in motor driving, and when he felt a little out of sorts he would get in his motor and drive forty or fifty miles, and, for the time being, that would throw off the obsession. But later, though he went to Paris on more than one occasion for a renewal of the treatment, his whole life became so dominated that he dreaded the sight of any dog, and the last time I saw him he was in a suburban private hotel, where he had a flat to himself, with two female nurses, and where his whole time was spent in watching lest any dog should approach either him or his room. And the nurses were, not infrequently, occupied for hours together in hunting to ascertain whether there were any hairs of a dog to be found on the carpet which might have been introduced into his food. By this time, of course, his whole life was so completely dominated and cramped by this idea that I recommended he should be placed in an asylum. At all events, I lost sight of him.

Similar cases have developed from the fear of syphilis, and recently, in a lecture I gave on "Syphilis and Insanity," I alluded to the condition which is called "syphilophobia," in which people who have contracted syphilis and people who fear that they may have contracted that disease, slowly become overpowered by the idea that they are suffering from syphilis, that they were possible sources, contagion-bearers, of syphilis to others, and they were altogether depressed.

One of the most interesting cases I ever remem-

ber, in which there was certainly no intellectual loss for a long time, was that of the son of a wealthy man who was trained at a public school and a university and who suddenly lost his fortune. The consequence of this was that he had to earn his living. His education at the University, where he had done fairly well, enabled him to get a tutorship, but he felt the change of fortune, and as it happened he was a man without beard or whiskers and with a rather Japanese aspect of countenance, he had the idea that people considered either that he was emasculated, or that he was a Japanese. He therefore avoided society. I may say he was highly musical, and for a time he found solace in his tutorial work associated with an out-pupil college, and his music. But the idea that he was looked upon as an alien so overcame him that he threw up all his work and became dangerous to society; he attacked one or two people, and finally he had to be sent to an asylum. I have known that man twenty-five years, during which time he has led an eccentric life, dominated all the time by the delusion that people looked upon him with suspicion, and yet, as I have said, earning his living by his intellect. Such a case may go on indefinitely.

Another case, almost as interesting, is that of a doctor who is still practising. He worked his way into the profession through a dispensership, and not only was he quite an efficient doctor, but he was also quite a good naturalist, an authority on some branches of English flora. Yet, with a con-

sideration and study of the occult, he developed a belief in hallucinations which he had, and for very many years he has been living what might be called a double life, i.e., a useful life as a general practitioner, trusted and looked up to by many—and I may say I have met him in consultation and found his knowledge of medicine efficient—yet, on the other hand, he is possessed with the idea that he is in communication with certain occult powers. You may say, "Well, but is not this the mere development of Spiritualism, and are you going to say that spiritualists have an invading mental disorder?" I should hardly say that, but I would go so far as to say that some spiritualists undoubtedly add their spiritualism to mental disorder, and that they develop it along the lines of such disorder.

One of the most striking cases I ever remember of the invading persisting growth which, however, did not affect the individual's mind, was that of a man who was in Bethlem Hospital when I was there half a century ago, and who was there twenty years later. He was a Civil servant, under the East India Company, and later he was attached to one of the outlying stations in India when the Mutiny broke out. He, with a few other Englishmen and some English ladies, were besieged in a small fort by a large number of natives. Definitely they decided that if once the natives got into the fort, each man was to shoot his womankind and then defend himself to the end. They were relieved, but that dominant idea that he must kill

his wife persisted, and, year in and year out, this man was in Bethlem Hospital doing quite useful work. He was an artist to a certain extent, and did a great number of illuminations for the hospital. Yet never could he lose the idea when he saw his wife that he must murder her.

Another line that I have described elsewhere is that in which you may say a normal growth of suspicion may pass into insane jealousy. The most striking example of this I think I ever remember was that of a woman, also in Bethlem Hospital, who, having been a dressmaker, became engaged to an artisan plumber. They decided that they could not marry yet, and that they must wait for a time. Finally, the journeyman got a little shop, where they had two extra rooms, so that, besides the shop, there was the little parlour-kitchen and their bedroom. And there the plumber worked, and worked strenuously. They decided they had better not have any children, and no children were begotten. Success attended the man's industry, and they were constantly talking about the time when they would be able to get a little suburban villa somewhere in the south-east of London. He continued to work hard, and at last determined he could carry this wish into effect. And, quite quietly, he got a small house in one of the suburbs, and got it furnished as well as ever he could, intending to make it a surprise Christmas-box for his wife. Everything was in order, everything satisfactory, and the happiness of the wife in her new little home can only be imagined.

Further success attended the man's efforts, and he had more than one shop, and had to procure assistance. Instead of being able to go to his little suburban home for a midday meal, he had to get a meal in the City, just getting home in time for an evening meal, and generally tired. His wife, no longer having to do all the housework as she had a servant of her own, found that time hung heavily on her hands. And her husband no longer coming home for a mid-day meal made it still more trying for her, and she began to wonder and wonder what could be detaining him. Then she got suspicious, and began to wonder whether he always came home the same way. Then she took to watching him, and came to the conclusion that he did not come home the straightest way. Finally, she made up her mind he must have some other woman whom he visited. This became so overpowering that she could resist the temptation no longer, and when the man returned one night he found his pretty house ruined; the wife had destroyed all the beautiful furniture, and had painted with ink on the wall the words, "Go to your whore!" In fact she fully developed delusions of suspicion arising from jealousy. The whole tragedy was manifest in such a case as that.

So I have described to you these invading morbid mental growths that, after all, have left little or no actual deterioration on the mind.

Now I have to pass more to the destructive or malignant mental growths. These, of course, are represented by a very large proportion of the

feeble-minded and demented cases met with in a large asylum. There is, to begin with, the large group of cases of so-called dementia præcox, in which youth, buoyancy of feeling, elasticity and well-being has been followed by an exaggerated egotism, followed by a maniacal condition, often associated with lust, with extravagance, and the like. Slowly this exaltation has passed into feeble-mindedness. In fact, every variety of dementia may have started with some comparatively slight mental disorder. The inevitable term "neurasthenia" crops up, and one is obliged to say that not infrequently has one seen the simplest case of neurasthenia develop into the most destructive form of mental disorder. Again, with adolescent insanity one has seen with fair frequency cases that could only be called hysterical pass from the hysterical stage into the maniacal stage, and the maniacal stage has been definitely and distinctly followed by evidences of mental decay.

Then come what are probably some of the most marked cases, the so-called paranoias. There is, for instance, the individual with the suspicion that spies are everywhere. There is the woman who has been left a widow with limited means and who feels she has to live a rather narrow and restricted life, and who accordingly isolates herself and then thinks that people are shunning her. Slowly she develops the idea that people are suspecting her of immorality. In one such case I remember seeing a woman who had gradually retreated up-

wards: from the ground floor she removed to the second floor, and when I saw her she was living in the attic, and even there she had plastered up the windows lest people should watch her. She felt quite sure that they accused her of immorality and of all kinds of sexual vices. I have seen a very large number of cases of this kind.

Another very troublesome group of cases are those of persons who think their doctor or their clergyman, who has been kind and considerate to them, is in love with them; and I have had, one may say, some amusing examples of unfortunate clergymen and—I was going to say—still more unfortunate doctors, whose domestic life has been entirely destroyed by the love entertained for them by some single woman.

Another group, of course, are those of people who think others are wanting to get possession of either them or their money, and in former years I used to be constantly hearing of cases of people who went to Scotland Yard to make complaints that they were being watched and followed. And it is very interesting to recognize how readily one may believe oneself to be watched. As a lecturer, I used sometimes to say to students, "Well, if you have lost a button from your trousers, it is pretty certain that, if you do not recognize it until you get outside, you will be sure to find somebody whom you think is looking at the spot from which you have lost the button." It is like the case of the provincial Senior Wrangler who, on coming to London for the first time in his life, thought

everybody recognized in him the distinguished Senior Wrangler. So a very simple real fact may give rise to ideas of persecution. Some of the most troublesome medico-legal cases I have ever had to deal with have been associated with some such disorder. In a famous case a certain lady believed that there was a conspiracy against her. She would engage a lawyer to protect her, and within a few days she would engage a detective to watch the lawyer. I have known what might be called a chain of detectives, each watching the others, and even the Judge before whom the case was to be tried was watched. I remember an interpretation she gave: she was quite sure that his judgment was not biased because she saw him standing under the three balls of a pawnbroker's shop, doubtless not decided as to which course he would take!

Such cases are, as I say, malignant in their development, the individuals, in many cases, becoming chronic lunatics.

It is not necessary for me to enlarge here upon the gradual growth of delusions of a destructive nature, as met with in cases of general paralysis of the insane.

And now, before concluding, I would like to refer to the possibilities of the utility of psychic analysis. As a rule, reasoning with the insane is a waste of time. You are told by Solomon you may answer a fool according to his folly, or you may answer not a fool according to his folly. In the majority of cases a person with a delusion is

unreasonable, and so reason, like water on a duck's back, has no effect. But that it may have effect was well shown by the following case :—

The son of a military man declined to follow his father's profession and took to science. Finally he took up electricity, and obtained an appointment connected with a South American cable. There he was leading a rather isolated life, surrounded by people whose language he only imperfectly understood. Slowly he developed the idea that they were not his friends, then that they were definitely against him and would like to get rid of him, and, later, he had the idea that all this followed as a consequence of his father's disapproval of his not entering the Army but taking up electricity instead. Then arose a very definite delusion, namely, that his father paid spies to rid him of his post in South America. Then came the most marked delusion, that his former tutor at the public school had been bought to come out to South America to cause trouble, that, fearing he might be detected if he appeared as the old tutor, altered his physiognomy and his general appearance so much that, although the young fellow was perfectly certain that a particular European there was his tutor he dare not tax him with the fact; but as this man disappeared in a short time he was convinced he was the tutor who had been spying on him. This led to inefficient work and to the man being sent home as a lunatic. He was admitted into Bethlem, and a more obnoxious and trying patient I have rarely had. He was perverse

in every way, so that when asked by an attendant or nurse to do anything, he always did the reverse. In the morning he would leave his bedroom in the utmost disorder on purpose to give trouble, and at meals he would cause much mess and spill things on the cloth; in fact, he was cantankerous to a degree. I talked to him several times, spoke of the absurdity of the idea of his tutor being sent out in the way he alleged, and I said of him, "If I got a letter from your tutor, would you be satisfied?" and he replied, "That would depend." I did get a letter from him, but he tossed it contemptuously on one side and said it was made up by me, and he did not believe it. I then wrote to the tutor, and said, "If by any chance you are in London, I wish you would come and see him." The tutor replied that during the Long Vacation he would come. And he did come and had a talk with the patient. He came away rather dissatisfied, and said, "I will see him again to-morrow I have promised to take him out to luncheon." He saw the patient the next day, and took him out to lunch and brought him back again. On the next morning the head attendant of the ward came to me and said, "Mr. So-and-So wants to see you." I said, "Really? Why?" "Well, sir, I don't know what has come on him, but he is an altered man; he made his own bed, and has been wanting to help us in every way; in fact, he has been as amiable and as charming as a man could be." Naturally I had him down to see me, and he said, "Doctor, I have got to apologize."

I said, "Why?" He said, "Well, I have been subject to delusions now for months." I said, "And now?" He replied, "I cannot understand it, but I know they were altogether wrong." I said, "Therefore you feel that you have been ill?" "Yes," he said, "and I am ashamed of it." I said, "You need not be ashamed of it, it is a question of illness." That man was discharged well. So here was a case in which, somehow or another, getting to the foundation of the cause of the delusion was followed by the delusion clearing off; and I have no doubt that, in psychoanalysis, there are conditions that are paralleled, from the salvation point of view, by conversions.

PYORRHŒA ALVEOLARIS AND ITS RELATIONSHIP TO DISEASE.

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I TRUST that the title of to-day's subject has not raised anticipations in your minds of a polemical discourse on the local pathology and local treatment of pyorrhœa alveolaris. These matters are the province of one's dental colleagues. I wish to take up the story at a later stage and, ignoring the origin and local manifestations of the established disorder, pass on at once to the secondary effects produced in *other* parts of the body.

The results of direct spread of infection from the tissues immediately around the teeth, important though they are, need not detain us long. Local abscesses, enlarged lymph glands, cellulitis, and various grades of septicæmia are familiar sequelæ; and the possibility of damage to distant structures, such as the joints and kidneys, by bacteria and toxins absorbed directly from the gums and disseminated by the blood-stream is realized and credited all the world over. To-day, I ask your attention for the bacteria which are *swallowed*.

The questions which require our answers are :—

(1) What is the fate of these organisms?

(2) Do they exert any harmful influences? And, if so, in what way can they be eliminated?

The number of different strains of micro-organisms present in the pus and debris around the teeth in pyorrhœa alveolaris is very great. The majority of them are saprophytes, preying on the refuse and food which collect between the tooth and the gum. A few, however, are infective, and grow in the living tissues around the teeth as well as in the discharges of the surface. Most prominent amongst them are streptococci; and I propose to deal almost exclusively with the effects of ingestion of these bacteria.

There is no doubt that the action of normal gastric juice in sterilizing ingested material is very great. At laparotomy I have often taken washings from the duodenum by means of a syringe and found them completely sterile. During various phases, however, of digestion or of indigestion, many bacteria escape. I have found them in the duodenum in large numbers during acute throat infection. They are often carried through to the jejunum embedded in protective masses of food. They survive also when gastric secretion is depressed, as by emotion, unappetizing food, during fevers, or as the result of chronic gastritis. To make a long story short, the assertion can be made definitely that streptococci of infective type do enter the intestines alive, and in many individuals establish a chronic infection in the chyme and in the mucosa.

Their distribution is peculiar. Table I represents one of a long series of cases in which the distribution has been studied. The figures denote the numerical relationship between the long-chained streptococci and the coliform organisms found at various levels during life.

TABLE I.

Showing the transition from Streptococcal dominance in the upper part of the alimentary tract to B. coli dominance in the lower zones.

Site	Ratio *Streptococcus longus* : *B. coli*			
	Case 1		Mean of 21 cases (81 specimens)	
	S. longus	*B. coli*	*S. longus*	*B. coli*
Mouth	100 :	0	100 :	0
Duodenum	... ...	...	100 :	0
Upper jejunum ...	100 :	0	100 :	0
Upper ileum ...	... ...	...	80 :	20
Middle ileum ...	... ...	...	59 :	41
Lowest ileum ...	99 :	1	46 :	54
Cæcum	16 :	84	24 :	76
Pelvic Colon ...	... ...	...	1 :	99
Fæces	0 :	100	5 :	95

In the mouth, duodenum and jejunum the infection is usually free from admixture with *B. coli*. The proportion of *B. coli* increases steadily at the lower levels and frequently is present to the complete exclusion of *Streptococcus longus* in the pelvic colon and fæces. In such instances the gums are infected with streptococci, the fæces with *B. coli*, and the intervening intestine with various combinations of the two bacteria. The zone of most intense infection is almost always in the lowest coils of ileum.

The formation of this mass of infected tissue is

of very frequent occurrence. The area of mucosa involved is many feet square. If bacteria can pass from it into the lymphatics and blood-vessels and thence to all parts of the body their opportunities for producing harm are very great indeed. I have investigated this question with some care, and have found streptococci in the lymphatic glands of the abdomen, in the peripheral blood-stream, and on occasion in the urine also, identical in every way both culturally and serologically with streptococci grown from the intestinal contents of the corresponding patients.

The clinical conditions in which this streptococcal invasion has proved to be most prominent are: chronic fibrositis of the muscles, arthritis deformans, chronic Bright's disease, many instances of minor and major anæmias, the chronic cardiac type of infective endocarditis, and certain forms of chronic colitis, but doubtless it plays an important part in the pathogenesis of many other disorders.

It is a matter of common observation that these conditions sometimes occur in patients whose teeth and gums are healthy. Patients with pyorrhœa alveolaris swallow streptococci in large numbers, but patients with healthy teeth often swallow them also, from other sources, such as infected tonsils or contaminated food, and so acquire chronic infection of the bowel with its attendant diseases. The converse sequence also occurs, and many patients with pyorrhœa alveolaris appear to escape the more serious forms of colitis and systemic

invasion because *Streptococcus longus* cannot establish itself in *every* bowel. It requires a certain combination of pathological conditions to enable it to gain a foothold, and these must be understood before logical treatment is possible.

Apart from a few instances of metastatic infection the whole gastro-intestinal flora is descended from organisms taken in through the nose and mouth. Possible but unproved exceptions are the motile bacteria which might conceivably ascend through the anus. The stomach and intestines of the newly born infant are sterile, but become infected with a rapidity which is remarkable. Germs in great variety are swallowed, but only such become conspicuous as find conditions in the intestines favourable for their growth. The composition of the chyme in breast-fed infants is very constant, and therefore the bacterial picture is constant, the number of races present being very restricted in spite of the diversity of organisms ingested. With additions to diet, environment in the intestines is modified and new races secure partial dominance. Progressive transitions take place until the flora of the healthy adult is evolved. An equilibrium is established; each bacterium, its chemical products, toxins, and ferments acting and reacting in concert with, or opposition to its fellows and their products. A complex series of balanced biochemical reactions is set up which can only be changed through alterations in local environment. Thus alterations in diet and assimilation modify the food supply, alterations in motility allow de-

composition to continue for a shorter or a longer time, and each change disturbs the original balance, substituting a new one in which the nature and relative dominance of the various bacterial strains are different. Organisms swallowed with the food and introduced into this cycle of bacterial life cannot reproduce themselves and thrive unless they are able to adapt themselves to their new surroundings. As an actual example, I may instance the repeated taking of sour milk bacilli. The sheer mass of bacteria ingested makes itself felt and modifies bacterial life in the intestines, but when the treatment is ended the intestinal flora quickly reverts to its original state, which reflects the nature of the environment offered by the gut. Hence it is that in many individuals streptococci pass into the duodenum alive, but their progeny is negligible and the strain quickly dies out. For the evolution of a state of chronic streptococcal infection the right kind of streptococcus must be swallowed by the right kind of patient. An analogy may make my meaning clearer. Let the soil of the countryside represent the contents of the intestines, and a handful of mixed seeds the bacteria. On the shallow chalky soil of the South Downs the crop would always be the same, short grass and gorse would predominate. If the seeds were put in the clay, a beech wood might spring up. In either place the seeds of tropical plants would have no chance to grow, although in the Indian jungle they alone would survive. The nature of the crop from a mixed

sowing is the product of the seed, but also of the climate and the soil.

Roughly two groups of streptococci succeed on occasion in establishing mucosal infection in the gut.

The first group are highly infective and make use of their great power of tissue invasion to escape from their saprophytic enemies in the chyme as quickly as possible. Swallowed in large numbers they cause acute bowel infection. Swallowed in small numbers they only survive in such patients as present zones of defective tissue resistance. For example, they may enter the tissues from a chronically distended cæcum, or a pelvic colon with stercoral ulcers, or a rectum affected by piles.

By far the more common and important group is the second one, composed of cocci of the lower degree of virulence but with strong saprophytic powers of such a kind that, given the right conditions, they may oust even the *B. coli* completely from large tracts of intestine. These may be termed glycophilic or sugar-loving streptococci. One is apt to think of *Streptococcus longus* as a somewhat delicate organism growing slowly under laboratory conditions, and these glycophilic cocci are no exception to the rule, except in the presence of sugar, when they grow even more rapidly than *B. coli* itself. Moreover, in sugary media they generate such a degree of acidity that no other infective organism can live, and even the robust *B. coli* dies out. The addition of only 4 per cent.

of these cocci to a culture of *B. coli* in sugar broth is sufficient to kill off all the *B. coli* in a few days' time.

TABLE II.

Demonstrating the Destruction of B. coli by glycophilic Streptococcus longus in the presence of sugar.

(The streptococci used in this experiment were isolated from the cæcum of a patient suffering from rheumatoid arthritis.)

Period of Incubation (Glucose broth)		Ratio *Streptococcus longus* : *B. coli*	
Expt. 1.	Hours	*S. longus*	*B. coli*
	0 ...	... 88 :	12
	12 ...	... 56 :	44
	24 ...	... 100 :	0
Expt. 2.	Hours		
	0 ...	... 13 :	87
	12 ...	... 34 :	66
	24 ...	... 72 :	27
	36 ...	... 100 :	0
Expt. 3.	Hours		
	0 ...	... 4 :	96
	12 ...	... 15 :	85
	24 ...	... 36 :	64
	36 ...	... 98 :	2
	84 ...	... 100 :	0

In Table II are shown the results of an experiment of this kind.

Infected gums are a source of such organisms and sometimes yield them in large numbers. Streptococci infecting the gums find themselves laved in a sugary medium derived from food debris of the pockets between the gum and the teeth and are gradually educated in the process of utilizing the sugar for their growth with the generation of acid.

Clearly the conditions in the intestine which

are necessary for the survival of this class of organism are stagnation and starch dyspepsia. Defective digestion of starch allows rich carbohydrate residues to reach the ileocæcal region. Stagnation, especially chronic ileal stasis, affords the cocci an opportunity to collect and so decompose the carbohydrate as to generate in places an acidity inimical to the growth of *B. coli* and its allies. When these conditions persist a chronic infection is produced, first in the chyme and then in the mucosa, and the way is prepared for systemic invasion.

Treatment.

The general principles for treatment are now evident. The infection in the mouth must be cleared up so that the massive action of the swallowed cocci may be abolished, and the uncontrollable modifications in specific resistance caused by the irregular discharge of toxins and cocci from the gums into the lymphatics and bloodstream eliminated. In the majority of cases evidence of continued invasion is patent even after the mouth has been cleansed, because the bowel infection when once established continues in only a slightly modified form even though the ingestion of infected material has been brought to an end. This is illustrated by the following case :—

R.M., a woman of 29, had suffered from constipation for many years and from a slowly spreading rheumatoid arthritis of seven years' duration. Such septic teeth as she had were removed in 1914.

Her tonsils were removed in 1916. During 1917 and 1918 repeated swab cultivations were made from the throat and gums, but no growth of *Streptococcus longus* could be obtained. Nevertheless her joints continued to swell, fresh joints became involved and she was clearly still suffering from active and increasing joint disease. In June, 1918, her colon was removed by Sir Arbuthnot Lane, and I made numerous cultivations at various levels of the intestines. The lower coils of ileum down to the ileocæcal valve contained a pure culture of sugar-loving streptococci. The cæcum contained streptococci and *B. coli* in the proportion of 21 cocci to 79 bacilli, whilst in the pelvic colon there were only 7 cocci to every 93 bacilli.

She is now free from arthritis and has had no swelling around her joints for about two years; but the point upon which I wish to insist most particularly is the evidence of severe bowel infection at a time long after the thorough cleansing of the mouth and throat.

Eradication of the bowel infection calls for patience and skill. A thorough survey of the alimentary tract is essential in all cases. In particular a detailed radiographic research into its anatomy and motility should be made and an analysis of the gastric content secured. The mechanical and digestive functions of the stomach and intestines must be raised to the highest degree possible, with the object of getting rid of all foci of stagnation where streptococci can multiply, and of preventing undigested food from reach-

ing the ileo-cæcal region for the nurture of these organisms.

Treatment must be adjusted to the needs of each individual patient. Its details are dictated by the findings of the preliminary investigation. When constipation is troublesome, I usually prescribe an aperient such as cascara, as well as paraffin, a combination of lubricant and stimulant being much more efficacious than the use of either drug separately. Irritant purgatives, massage and active exercises should be employed with care. Until the streptococcal infection has been got well in hand, these measures, by stirring up the infected area do more harm than good. The increased dissemination of cocci and toxins which they occasion aggravates all the symptoms and is very detrimental to the patient.

Of electrical methods, I find it best to start with the less vigorous varieties first. Abdominal diathermy, which by heating up the mucosa and muscle coats of the gut, promotes peristalsis, and the flow of blood and lymph is of the greatest use. An application of $\frac{3}{4}$-hour duration should be given 3 to 6 times a week. At a later date more stimulating methods can be used, especially surging galvanism, a slow rhythmic rise and fall in potential being a most effective means of exciting regular peristalsis.

A well-balanced, simple, mixed diet is the best, and one in which particular attention is given to the starchy foods. These should be taken in limited quantities and only in their most digestible

forms. The diet should be appetizing, so that an efficient psychic flow of gastric juice is evoked. Unpalatable diets, although made up of foods which are readily digestible in the laboratory, often prove most indigestible in the alimentary tract. The disgust and ennui which they cause hinders both the flow of ferment juices and the proper peristalsis of the gut.

Any defect in stomach acid should be made good, and some powerful starch-splitting ferment given to hasten the digestion of the starchy elements which are especially harmful. I have found taka-diastase given in doses of $2\frac{1}{2}$ gr. at meal-times very useful for this purpose. These measures are designed to modify the environment of the bacteria in such a way as to render it unfavourable for the survival of streptococci in the chyme, and it is only by methods of this kind that the intestinal flora can be modified appreciably. In Table III is shown the effect of restriction of the intake of starches on the fæcal flora.

The action of intestinal antiseptics in the same direction is notoriously fickle. I find, however, that colloidal sulphur and a combination of ammonium ichthyosulphonate with proteins are of the greatest service to many patients.

The task of destroying the cocci which have entered the tissues still remains for consideration. *General resistance* should be raised by the treatment of collateral disorders, such as anæmia, and by securing healthy and restful habits and surroundings. As I have already mentioned, the

early weeks of treatment must be passed very quietly. Activity merely aggravates the condition. The endocrine glands are often damaged by the infection, and a higher level of general resistance can be attained by the administration of glandular extracts which supplement the deficient secretions. Thyroid extract is of particular importance.

TABLE III.

Demonstrating the diminution in Streptococcus longus in the faeces which follows the elimination of carbohydrate from the diet.

Day of Observation	Diet	Ratio *B. coli* : *Streptococcus longus*		
		B. coli	*S. longus*	
1	Full	100	15	Mean 17·3
2	„	100	16	
3	„	100	25	
4	„	100	13	
5, 6, 7	Carbohydrate—free			
8	„ „	100	3	Mean 1·7
9	„ „	100	1	
10	„ „	100	0·7	
11	„ „	100	2	

Many of the measures already described for dealing with infection in the chyme are also useful agents for increasing *local resistance* in the bowel. The elimination of intestinal stasis and distension and the promotion of blood and lymph flow by abdominal diathermy are perhaps the most important. Local resistance should be increased in other places also, wherever metastatic foci of infection have established themselves. For example, resistance can be raised locally around infected joints by means of a radiant heat or baths of paraffin wax.

Lastly, the patient's *specific resistance* should be raised to the highest degree possible by vaccines. In the preparation of these vaccines strains of streptococci from the *intestines* should be used whenever possible as well as those from the gums. The biochemical characters of the two classes are different, owing to the different conditions under which they have evolved. Here a serious difficulty arises. Extensive streptococcal infection can, and, as I have proved, often does exist in the small bowel even when the fæces are free from these organisms. It is sometimes impossible to obtain any of these strains apart from laparotomy. The best way of dealing with cases of this kind is to examine the fæces after a course of stimulant purgatives, such as aloes or cascara, given for two or three days in succession, and if streptococci do not then appear, examine the fæces again after a course of lactose and magnesium sulphate. This combination, given for several days before breakfast, flushes the colon with sugar, and causes the streptococci to increase in number, so that at last they appear in the evacuations.

Summary.

Some of the chief points of clinical importance are :—

(1) Streptococci swallowed in the pus from pyorrhœa alveolaris frequently infect the bowel, its lymphatics, and eventually the blood-streams and distant tissues.

(2) This infection persists long after the mouth is cleansed.

(3) This infection often exists even when the fæces are free from living streptococci.

(4) The evolution of many cases of this chronic bowel infection is determined by the co-existence of chronic intestinal stasis and of starch dyspepsia.

The evolution of others is determined by the presence of foci of depressed tissue resistance as in a distended cæcum or congested rectum.

(5) Treatment should include :—

(*a*) Cleansing the mouth.

(*b*) Altering environmental conditions in the gut by :—

(i) Correcting disorders of motility.

(ii) Correcting faulty digestion, especially of starchy foods.

(*c*) Raising general resistance.

(*d*) Raising local resistance.

(*e*) Raising specific resistance.

(6) The disease is a chronic one. It is evolved slowly and requires prolonged treatment; but if the preliminary investigation is thorough, every patient can be greatly benefited or cured.

DISABILITIES OF THE FEET DUE TO STATIC OR MECHANICAL CAUSES.

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In the first of these two lectures I propose to deal with those structural and functional defects of the feet which result either from purely mechanical weakness, or from minor conditions of injury and disease whose mechanical effect is more obvious and of greater importance than the causative lesion itself. The second lecture will be devoted to deformities and disabilities produced by gross paralyses of one or more of the groups of muscles below the knee. To illustrate my points, I have brought here some cases showing typical conditions, and others the results of treatment; still other deformities are represented by this series of plaster casts.

It must first be realized that the foot serves two quite distinct purposes, and that its shape and use in these two functions should vary accordingly; the one is for standing and the other for walking. The former is a mere "pedestal" action, or one of passive support of the body weight, in which

muscular action is reduced to the very minimum necessary to maintain balance, while the ligaments are stretched and tight, and the various joints of the foot forced into an extreme position and "locked" to convert the otherwise supple foot into a rigid whole. In this weight-bearing action all the arches tend to be slightly lessened, not by stretching of the normally unyielding ligaments, but by the gliding of the normal joint surfaces. The second purpose of the foot is that of locomotion, assisting the leg in the forward propulsion of the standing body. Here the foot acts as a machine, although a simple one, and characterized mainly as a lever of the second order. Contrasted with the passive weight-bearing action, we now find that ligamentous strain is reduced to the minimum, whereas the muscles are in full action. Ligaments are slack, joints are unlocked and tend to fall into their "middle" or easy position, arches are restored or exaggerated, balance is complete and poise graceful, and the muscles are in full tone, engaged in, or ready for instant, action. The appearance and shape of the foot, according as the foot is acting as a pedestal or as a muscular organ, is known as the "attitude" of *rest* or *activity*. Care must be taken to distinguish between the meaning of the term rest as here applied (i.e., the weighted foot when the body is "standing easy") and that denoting absolute rest —the unweighted foot when sitting or lying. With the latter we have nothing to do, except to say in passing that the shape of the foot here resembles,

in the formation of the arches, that seen in the attitude of muscular activity in walking. This interchange or "alternation" of the shape or attitude of the foot from that of restful passive support to one of active muscular action, constantly occurs in healthy feet in getting about ordinarily, and is a wise provision to ensure adequate rest for the two important structures—the propelling muscles, and the strain-bearing fibrous tissues. Except in persons in good physical condition, the weaker element in this couple, and therefore the more liable to be exhausted quickly, is the musculature. When the muscles have given up, so to speak, it is still possible to walk about in an ungraceful way, shambling along with the feet stiff and locked, making the ligaments serve instead of the muscles, and throwing excessive work on the former. Again, in occupations necessitating standing about or standing relatively still, without periods of active walking between, the alternation is disproportionate, and, subsequent to the fatigue and aching of the muscles, we notice the same symptoms in the ligaments, combined with tenderness and perhaps with acute pain and tearing sensations. Thus with misuse of the foot the alternation tends to be eliminated, and the patient walks in the passive attitude, using the ligaments instead of throwing the muscles properly into action. Many minor defects and disabilities of the foot, such as corns, scars, local arthritis, ill-fitting boots, and other causes of pain or discomfort, have a general

disabling effect on the whole foot quite out of proportion to the local damage, in that, to lessen the discomfort, the patient locks the foot and holds it rigidly or in some strained position, thus substituting only too readily the passive attitude of weight-bearing for proper muscular or leverage activity. The gunshot wounds of the foot seen in the late war showed most frequently how a seemingly trivial injury to one or other of the small bones or joints, although of very local extent, compelled the assumption of the passive attitude in walking, and rendered the man quite lame or considerably disabled. The want of alternation of attitudes has a vicious effect, in that the constant use of rigid joints and a stiff foot, to the exclusion of muscular action, leads more and more to muscular atrophy and insufficiency, to fixed habits of improper walking, and finally to structural alteration and fixation in the bones, joints and ligaments. The ligaments and plantar fascia are inextensible under normal conditions, and yield only so far as their slackness is taken up by the movements of the joints through their normal range; they therefore prevent subluxation of the joints. With over-use, however, they gradually stretch, and permit abnormal displacement of the joint surfaces, and thus deformity arises. Structurally the ankle and sub-astragaloid joints, like the knee, are weak, the weight tending to fall on the outer side of the joints; any yielding at the knee and "ankle" is in the direction of exaggeration of that slight degree of in-bowing which

normally exists (knock-knee or genu valgum, and "weak ankles" or pes valgus). It must be remarked here that the ankle and sub-astragaloid joints should be considered together as a combination permitting universal movement, but that the exaggerated projection of the inner malleolus ("weak ankles") is due to yielding in the joint beneath the astragalus, and not in the ankle-joint itself. The normal locking of the three large posterior joints of the foot—the ankle, sub-astragaloid, and transverse mediotarsal joints—for the purpose of weight-bearing as opposed to muscular action, is in the direction of dorsiflexion and abduction (eversion, pronation), and the most important fibrous structures are the deltoid, interosseous, and calcaneo-scaphoid ligaments, and the plantar fascia. The persistence of the passive or abducted attitude, instead of the regular alternation with that of plantar-flexion and adduction which characterizes the active muscular attitude, or the exaggeration of the normal pes valgus position, is the essential sign of a *weak* foot, or one in which the function does suffer, or will deteriorate without care.

The term *weakness* is not to be confounded with *flatness*. A better conception is obtained by realizing that the majority of cases of acquired flat foot were in the beginning not flat foot (pes planus), i.e., were not due primarily to stretching of the long arch of the foot, but began as cases of abducted foot (pes valgus), resulting from the incorrect persistence of one of the two physio-

logical attitudes, and characterized by a tendency of the leg and astragalus to roll in and rotate in, in a subluxated position over the inner border of the foot. The os calcis topples over towards the inner border, and the weight line falls nearer to, or within, its inner tubercle. The head of the astragalus is depressed and projects on the inner border of the foot. Later, strain falls on the transverse mediotarsal joint, and that part of the foot in front of this joint becomes abducted and dorsiflexed relative to the posterior half (astragalus and os calcis). The fore-foot takes an everted or abducted twist, counter to that of the leg and back-foot. It is in this later stage of yielding of the sub-astragaloid joint that falling of the long arch, or planus, results from the initial valgus; but the sequence is far more characteristic of adults than of children, in whom, as a matter of fact, acquired planus is very uncommon, while pes valgus, or "weak ankles," is very frequent. When the shape of the unweighted foot is compared with that of a foot bearing weight, it is noticed that the normally concave inner border becomes straight or slightly convex on standing, while the fore-foot becomes everted; also, dorsiflexion occurs at both ankle and transverse tarsal joints, and the long arch is slightly flattened. Again, the transverse arch of the metatarsal heads and that across the mid-foot also tend to be lessened. Thus it is seen that a certain degree of yielding is normal, but only so far as the unstretched ligaments permit the joints to glide.

Exaggeration of this motion, due to misuse of the ligaments, produces deformity and structural weakness, though not necessarily disability.

Whether a deformed foot is also disabled, providing in either case the muscular power is not primarily at fault, depends on the degree of movement of its joints; and it may be affirmed that the first essential in treatment is to restore completely the range of movement of all the joints of the foot, including the ankle, so that they can be actively and painlessly carried out by the patient. Whether this can be done depends on the nature of the restriction; a foot stiff and painful from the presence of adhesions may be quite cured by thorough manipulation under an anæsthetic, while long-standing rigidity with mechanical articular changes may be made much worse by mobilization treatment. In cases of pure flat foot (planus) of inherited or racial origin, the foot is very mobile, but the complete adaptation of the bones and joints to function through long-continued use, under efficient muscular control, renders the foot functionally perfect, although flat as judged by other standards. In children we frequently find weak feet, but no painful disability; with these, however, there is often a weak and ungraceful gait. A considerable degree of deformity may occur, with much stretching of ligaments, but it is rare to have children complain of pain in the feet from this cause; this is due to a greater adaptive facility than obtains in adults. Children's ligaments never have been developed strongly, or stretch so

gradually that no pain is caused. In adults we are much more liable to find the slightest degrees of deformity, or even none at all, associated with a quite painful disability. The undue strain on the calcaneo-navicular and deltoid ligaments, and on the plantar fascia, soon shows itself in acute pain, sometimes with a tearing sensation, and associated with tenderness on pressure. There may or may not also be effusion and tenderness in the synovial tendon sheaths, and swelling and congestion of the affected ligaments. Later, some measure of deformity, often slight, may occur, and the pain on movement compels the patient to keep the foot rigid, and walk in the attitude of rest or abduction. In some cases, especially in adolescents among the working class, reflex spasm of the peronei and dorsiflexion muscles adds greatly to the fixity. Walking on locked joints, fixed in their extreme positions and meant only to be still, gives rise first to synovitis and arthritis, and later to alteration of the joint surfaces, denudation of cartilage, and osteophytic formation, with further fixation, pain, and the other results of traumatic or mechanical arthritis. Adhesions occur both in the tendon sheaths and joints, and the absence of muscular movement, in addition to atrophy from disuse, leads to eversion contractures and to the weakening over-stretching of antagonists. With the want of muscular action, the swelling and congestion due to vasomotor effects is added to that of the inflamed tissues.

It has been said above that the foot in its active

muscular function is a machine. It follows, therefore, that its mechanical efficiency is governed by factors determining that of an inanimate machine, and thus the static causes for its breakdown are readily realized. The anatomical structure has already been dealt with, and it remains to consider the amount and method of the work of the foot in relation to its strength. In propelling the body, the foot is a lever whose fulcrum should be the five metatarsal heads and the toes (although only too often it is incorrectly formed by the first and second metatarsal heads and the inner side of the big toe); the resistance or "load" is exerted through the leg and astragalus; and the power is applied to the heel by the calf muscles, assisted by the flexors of the toes, tibialis posticus, and peroneus longus. It is important to note that the movement at the ankle-joint is not one of dorsi- and plantar-flexion in the plane of the leg, but that dorsiflexion is combined with marked abduction or "valgus," and plantar-flexion with adduction or "varus." The action of the three main rear joints of the foot cannot be dissociated; adduction in the sub-astragaloid is added to plantar-flexion at the ankle; plantar-flexion, adduction, and rotation, in the transverse tarsal joint are combined with the adduction and plantar-flexion in the other two big joints. The question of the plane of movement in the leverage action of the foot is of importance in the decision of the vexed question as to what height the heel of a boot or shoe should be made, to give comfort in walk-

ing, and relieve the foot from undue strain while not lessening its efficiency. The higher the heel, i.e., the more the plantar-flexion, the greater is the adduction of the foot, and therefore the less the strain on the inner ligaments and on the inner tibial muscles. A raised heel, by producing a varus position, tends to unlock the sub-astragaloid joint, as the latter is most fixed when the heel of the foot is on the ground and the foot in valgus position. By the raised heel of the boot the joints of the foot are thrown into "middle" or easy positions, and the locking stability of the foot is in a certain measure sacrificed to ease ligamentous and muscular strain. Besides furnishing ordinary protection and warmth, the essential use of a boot, developed as a result of custom, is to protect the ligaments from over-strain. In plantar-flexion, as in the use of a high heel, the curvature of the long arch is increased, and a good shoe should accordingly have a well-curved and stiffened waist. In such a shoe the weight-bearing surface of the foot is greater and more generally distributed, and all the ligaments supporting the long arch and the inner ankle are thereby relieved. The more the heel is raised the less becomes the amplitude of plantar-flexion leverage action, and the shorter the step taken; so the heel-raising remedy for weak feet must not be carried to an absurdity. Besides being high, it is essential for the stability of the shoe, and therefore of the foot, that the heel be as broad as possible; and as, from a cosmetic standpoint, height and breadth of

heel are not consistent, in a remedial shoe height must be kept within reasonable limits by considerations of breadth and support. The height of heel supplied to women for remedial purposes should not exceed two or two and a quarter inches, and in house shoes it should not be less than one and a quarter. The maximum height given should be no more than affords relief, and in any case of overstrained foot heel-less slippers must be avoided. Patients should be instructed to put on the shoes immediately on getting up. With regard to the type of heel, the " Cuban " is the most satisfactory for remedial or even general use; the curved " Louis " heel is bad on account of its unmechanical shape and inefficient support, and because it weakens the whole sole of the boot. In a strong foot, with no contracture of the tendo Achillis, the lower " Brogue " heel of about three-quarters to an inch in height may be permitted. While there is no doubt that some women wear heels of excessive height for purely cosmetic reasons, it is equally certain that the habit of using a moderate height by the great majority of women is a response to the relief afforded to the ligaments by the adduction of the foot which the higher heel favours. It may be that it is at times overdone, as the result of custom, and the utility sacrificed somewhat; in this case the effect may be vicious, leading to permanent shortening of the tendo Achillis and necessitating further increase of the height of the heel, but lowering the muscular efficiency of the foot. The shorter steps necessitated by heels of

moderate height can be of benefit to the musculature of the lower leg and foot, in that more work must be done by the foot in locomotion and less by the hips. Women, being hampered by skirts, cannot get such exercise and freedom of movement in the hips and thighs as men can, and it may be from this cause that they need the more efficient support to the feet afforded by heels. The leverage action of the foot in men as a whole is not nearly so marked as in women; men propel themselves more with the hip movements of long strides, whereas women walk more with their feet. For the efficiency of the foot itself a short slow step is more valuable than a long quick stride.

The ligaments most liable to strain lie on the inner border and under surface of the foot, and therefore a relieving support should promote adduction (varus) as well as lift the foot. We have seen that a moderate heel and a strong, well-curved waist to the boot conduce to this, but distinct additional help is rendered by specially strengthening that part of the "upper" of the boot or shoe (known as the "golosh") which lies along the inner border of the foot between the heel and the big toe. The stiffening here, which in a specially made boot consists of an extra leather insole, supports the head of the astragalus, navicular, and cunieform, as well as the inferior calcaneo-navicular ligament; and it prevents the subluxation inwards, and the foot from breaking at the mid-tarsal joint. The upper should fit most snugly over the mid-foot (the "instep"), and the lacing should permit the

inner border of the foot to be braced up most firmly. Most ready-made shoes and boots fail miserably in the support afforded to the inner border of the foot, and this, together with waists that are thin and yielding, and which become convex downwards after a week or two's use, is largely responsible for the non-efficient ligamentous support and the breakdown of the foot. Where such an evil arrangement is combined with a "flat" heel, the shoe is of the worst, and it is no wonder that in any large collection of women workers, such as nurses, who are much on their feet, we see many cases of over-strain. A much used method of lessening foot strain by promoting adduction is that of "wedging," the inner border of the heel, or heel and sole, being thickened by the addition of extra leather to the extent of from one-eighth to one-third of an inch, according to circumstances. With this the heel is sometimes brought forward on the inner side, further to support the waist. Wedges are more useful in stoutly waisted boots, and where the heels are not high, and are therefore of greater service to men and children than to women, as, in the latter, wedging a high heel throws the ankle out too much and makes the foot insecure.

With the question of wedges should be considered that of "arch supports" (or valgus pads and plates). These unquestionably give relief in many cases, but the difficulty with the shop-made article is that of frequent misfit, as the supports are made to standard patterns, whereas there is

no standard foot. Many surgeons object to their use—I think unreasonably—the objection being that the support is alleged to exercise pressure on the intrinsic muscles of the sole, and thus cause atrophy and weakness. But in my opinion this allegation is untrue in the case of well-fitting supports made specially for the foot concerned, and fitted by the surgeon himself. Such a support has two extremely valuable functions. Firstly, it acts not by pushing up the arch and compressing the sole muscles, but as a wedge inside the shoe, extending from the inner tubercle of the os calcis to just behind the great toe joint, and serving more adequately the purpose of the wedges mentioned above which are attached to the sole and heel. Secondly, the support and its flange prevent the waist and the inner side of the shoe from collapsing with weight-bearing, which is so liable to occur in ready-made shoes, and thus enables the shoe to maintain itself as a continual support to the arch. Arch supports in ready-made shoes, transferable from one shoe to another, are just as efficient and a much less expensive form of treatment than ordering specially made shoes. Tender feet, however, may find them a little too rigid, but the objection of weight can be met by making them of duralumin or other light alloy. In general, the shop-made supports have been reduced to the minimum size; others of a type with an outside flange are too clumsy and heavy. Rubber pads and other such substitutes, except the leather insole made on the last of the

boot, are generally quite useless. In children, whose attention cannot be concentrated to maintain proper posture and correct walking, when a considerable over-correction of the foot is desirable, it is often found that the combination of an arch support with wedges on the sole and heel of the shoe is most efficacious.

In estimating the efficiency of a foot, regard must be paid to the extent of passive movement in its individual joints, and more particularly in the ankle, sub-astragaloid, and mid-tarsal joints. Any restriction of movement, in so far as it can be overcome permanently, should be rectified. The joints must be examined separately to gauge wherein the restriction lies. Stiff joints, unless absolutely ankylosed by bony union, are nearly always painful, or, if not, they at least conduce to deformity and bad function. The ankle joint should permit plantar-flexion until the dorsum of the foot is in the same plane as the front of the leg; and *passive* dorsi-flexion, *when the foot is somewhat adducted or inverted,* and the knee fully extended, should reach its limit only when the foot forms an angle of 80 or 85 degrees with the leg. This latter test of *passive* dorsi-flexion is most important, as only by this can be detected those slighter degrees of permanent shortening of the tendo Achillis, which so frequently cause abduction, and subsequent breakdown, of the foot. This contracture of the heel tendon, without any obvious cause, is most frequently met with in mechanically disabled feet, and at all ages. We do not refer

here to the numerous cases of heel contracture resulting from paralyses, prolonged recumbency, etc., but to those found in many cases of "flat feet" where there is no evidence of the original cause of the contracture. In children it is possibly congenital, or resulting from some overlooked neuritis in a transient infectious fever; at times it is associated with genu valgum and recurvatum and other signs of deficient muscular tone. In adolescents it possibly persists from infancy, but in other cases, such as long-standing severe plano-valgus, the contracted heel tendon may more reasonably be held to be the *effect* and not the cause of the foot deformity. Women who suffer from weakness of the feet, far more often than not have short heel tendons, and though in some cases the condition may have persisted from earlier life, yet it must be assumed that in many others the condition is caused or exaggerated by the habit of wearing the heels unduly high—higher than a requisite support would necessitate. At times the short heel, with resulting abducted foot, is met with in dancers improperly trained, when the calf muscles have been unduly developed. To overlook a short heel tendon in a case of "flat foot" is to make the usual remedies—boots, supports, massage and exercises, walking and standing drill—quite unsuccessful. A good test for short heel tendons is to ask the patient to stand with the feet parallel and well adducted or varus, (i.e., standing right over on the outer borders of the feet) and with the knees fully extended; if the tendons are short

the patient will be liable to fall backwards. The methods we adopt to lengthen the heel tendons will depend on the age and other circumstances of the case. Slighter degrees in young children will react to passive stretching, and to active exercises such as walking up an incline with the knees straight and the feet strongly supinated (i.e., on the outer borders). More resistant cases should be given a light metal night splint, with the foot piece strongly dorsiflexed and inverted. If it be desired, a quicker result will be obtained by a Bayer's tenotomy of the tendo Achillis, followed by plaster or splint for a couple of weeks; a lengthening of about half an inch will usually suffice. In adults, except in the severe cases met in hospital practice, it will rarely be necessary to have recourse to such a drastic measure as tenotomy, and in most cases it is sufficient to raise the heel of the shoe to that degree which gives comfort to the patient, and objectively throws the foot into a better position. In cases where the Achilles is short the treatment of the weak foot may be undertaken coincidently with that of the heel tendon, except in cases of preliminary tenotomy; but until the heel tendon is lengthened no structural improvement in the foot can be expected.

The sub-astragaloid joint, with which must be considered the mid-tarsal joint, should, in a good foot, have an inversion-eversion range of 80 or 90 degrees, and is important in balancing movements and in the completion of extreme plantar-flexion. Adduction is more liable to be restricted,

and should be restored. In advanced cases it is limited by adaptive contracture of the peronei tendons, ligaments, and other soft tissues on the outer side of the foot and ankle, consequent upon the persistence of abduction in improper walking, or from adhesions or spasm. Still more advanced cases are restricted from alteration of bony configuration, as laid down by Wolff, and these structural types are amenable only to such resection operations as that of Ogston, a measure rarely called for. Early cases of passive restriction of adduction may be the result of intra- and extra-articular adhesions following synovitis of the joints and tendon sheaths. In any case, the treatment is to examine the foot under an anæsthetic, and, by gentle manipulation in all directions, to break down any adhesions and estimate what adaptive contracture, if any, exists. Care must be taken not to tear ligaments or other tissues. In many cases of painful foot, where the condition is obviously not acutely traumatic or inflammatory, and where on ordinary examination there appears to be only the slightest or no restriction of movement, a gentle manipulation under an anæsthetic, with a stretching of all the joints in all their combinations of movements, followed up immediately by complete functional use, will give a marvellous result in freeing the foot from pain. When no contracture exists after the manipulation, and the foot is therefore quite free, immediate use is insisted upon, assisted perhaps for a few days by massage, active and passive exercises, and with

such foot support as is necessary. Impediments to restoration of movement due to contracture of tendons are met by tenotomy, as of the peronei and Achilles, and by division of resisting ligaments, followed by fixation in plaster or in a splint in the over-corrected position, for just such time as is necessary to stretch permanently those tissues which cannot be cut. The plaster stage is followed by a period in which physio-therapy and muscle re-education is practised, but weight-bearing is not allowed until the function is good enough to make relapse improbable. Eventually walking is permitted, but with the foot protected by boots, wedges, arch supports, and possibly also an outside leg steel and T-strap, the re-education proceeding the while. The walking supports and instruments should gradually be reduced as the foot is able to take care of itself. In advanced cases with severe planus and valgus, with contracture of the Achilles and the peronei, and with the mid-tarsal joint broken down, and in which the back-foot is really plantar-flexed, abducted, and inverted at its fore end, while the fore-foot is dorsi-flexed, everted and abducted, the correction may require to be done in two or three operations, it being impossible to correct the three big joints together at one sitting. But in such cases, besides the contracture of soft parts, there is usually a considerable alteration of the shape of the tarsal bones, and an advanced degree of mechanical arthritis, and these make the eventual functional result so poor that extensive correction is scarcely

worth while. It is better to give such cases those simpler measures which suffice to afford them some relief than carry out any radical procedure.

The transverse mediotarsal joint assists the sub-astragaloid by permitting the foot to swing into adduction about the astragalus. It permits also dorsi- and plantar-flexion of the fore-foot on the back-foot in the same oblique plane as in the ankle-joint. Again, it allows inversion and eversion of the fore-foot, and also a slight degree of rotation. These movements should be considered in manipulation procedures under an anæsthetic, and at the same time the scapho-cuneiform-metatarsal joints should be mobilized. It frequently happens that adhesions about these joints render them very stiff, and cause pain which is at once relieved by a good stretching. For such a local condition as this an anæsthetic is not always necessary.

The foot, like any other machine, may fail in its work either on account of its intrinsic weakness, or because its "load" is disproportionate to its strength. It may be called upon to sustain too great body weight, as exemplified by the numerous cases of disability occurring in women who grow stouter after the menopause. Or the work may be too much in the matter of length of time required in standing at some occupations. Standing is much more harmful than walking, as in the former the regular alternation of attitudes, the sparing of ligaments by muscles and vice versa, do not occur. Muscular and ligamentous strength, especi-

ally the former, may be deteriorated by general illness and enforced recumbency, and thus cases of foot strain are often met with in patients convalescing from illness. Bad hygienic conditions, such as poor health, insufficient food, long hours, laborious occupations in adolescents, and too much standing in children, also act by lessening the efficiency of the foot in relation to the work demanded of it. Existing deformity, even in a supple foot, unless racial or inherited, cannot but lower the working efficiency. Without doubt, the two factors which predispose most to the breakdown of the foot are : (1) Improper habits of walking and using the feet, and (2) ill-fitting footwear. The use of the boot, and the description of the conditions it should fulfil in regard to the long arch, have already been described, and an account of the toe portion will be given when discussing the anterior transverse metatarsal arch. Ill-fitting boots, by cramping and restricting joint action, by yielding and giving insufficient ligamentous support, and by producing corns and other minor disabilities, either directly injure the foot itself or indirectly have effect by compelling incorrect functional use, and preventing the proper alternation of attitudes. While the position in rest in standing is such that a little eversion of the feet is normal, the foot joints being then locked and the knees facing directly to the front, this attitude is incorrect in walking. In a correct walk, the foot joints are unlocked, and muscular support replaces ligamentous as far as possible. The feet are used in

parallel relation as regards the inner borders, except at the end of the stride, when the big toe should turn in towards the middle line, and the "push-off" be made by the balls of all five toes and the toes themselves. In the foot advanced for the next step the weight is first thrown on the heel, then rather more on to the outer than the inner border of the foot, and finally finishing on the toes as described above. The stride should be slow and short, as the shorter the step the greater the leverage action of the foot, and the less is locomotion carried out by hip-joint action. The sequence of the change of the weight-bearing point of the foot, if properly carried out, will compel a gentle toes and heel action. Such then, is the re-educative "foot drill" which must be carried out after contracture and rigidity have been overcome. It is usual to prescribe exercises, but it must be remembered that the training to produce a correct standing posture and proper habits of walking, carried on until correct function becomes automatic, is of far more importance than definite set exercises. The latter are supplementary, and may include certain exercises, free-standing or not, done with or without resistance, and not forming part of the action of walking. With them, too, are included heat treatment, baths (hot and contrast), passive movements and massage, and any other physio-therapeutic measures assisting mobility and muscle education. The exercises have as their principal object the strengthening of the inner tibial muscles, the flexors of the toes,

and the calf muscles, and may be done with or without shoes according as the foot is weaker or stronger. Running, dancing, and skipping form useful and strenuous adjuncts. While ordinary or ball-room dancing is most helpful in strengthening a weak foot, yet fancy or ballet dancing, because of the extreme eversion or abduction "positions" used, must be absolutely forbidden as a remedial measure.

There remain yet to be mentioned some further measures and results of treatment. The relation between the degree of deformity and that of disability in an overworked foot varies enormously, and depends largely on the age of onset, and on the duration of the condition. As a rule, in children we find very obvious deformity with no painful disability, while adults may suffer extreme discomfort when little alteration of shape can be detected. Pain in the ligaments and plantar fascia in adults, when no infective process is present, means progressive deformity, but it can be quite relieved by sufficient rest and the measures previously mentioned. Where the condition is very acute, and associated with swelling of the joints and tendon sheaths, or with muscular spasm, it may be necessary to take the patient off her feet absolutely for a week or two, giving massage meanwhile to assist the circulation and strengthen the muscles. If walking is permitted, it should be limited, and be done only in such boots as give maximum support. A mild attack of stretching and pain may be quite cured by temporary firm

strapping of the mid-foot and ankle-joint in a transverse and "figure of eight" method. Subsequently, the amount of weight-bearing and the kind of footwear must be controlled. No adult patient with a purely static foot condition need remain uncured, or at least not considerably relieved, but the cure can remain incomplete on many occasions on account of the conflict between what is necessary and what is expedient. Adult patients cannot be expected to submit to ideal operations for conditions which do not shorten life, and in orthopædics, as in other branches of surgery, it is just as necessary to treat the patient as to treat the feet. It must happen at times that the compromise is unsatisfactory. Cutting the heel tendons, or giving ugly "surgical boots," are measures to which the majority of adults will not submit; raising the heels and giving a pair of arch supports will often be far more welcome. Flattening of the arches (planus), if the feet are supple and free from pain, is of no practical importance to the patient; the condition may require only such boots or supports as will keep the foot in the best position for doing its work, and prevent increase of deformity. Mere flattening of the arch unaccompanied with disability, and when it has existed a long time in adults, cannot be cured except by an extensive operation to shorten ligaments and tendons, and for this purely cosmetic purpose it is not worth while; not all the physiotherapy and muscular re-education in the world will serve to reduce chronically over-stretched ligaments

to their pristine length. Pes valgus, or abducted foot, can be held in check by wedges, arch supports, and heels, and discomfort is thereby averted. With children the outlook is somewhat different. Provided there is no Achilles contracture, the use of wedges and supports, together with thorough foot drill, may restore a flattened arch to its normal, and I have seen bad cases of deformity completely corrected in so short a period as three months. In children, however, we meet with valgus deformity ("weak ankles") far more often than we do real flat foot (planus); in fact, the latter condition is quite rare. Valgus in children may take years to improve, but the delay is often due to slackness of control on the part of the parents, and the want of assiduity with which the prescribed treatment is carried out. It is probable that many of the slighter cases of "weak ankles" tend to grow out of the condition in time, in spite of the absence of treatment. On the other hand, however, many cases of valgus do not improve spontaneously, and as a later result flattening of the arch (planus) supervenes on the valgus. In these severer cases in children (from about the tenth to the fifteenth year), in which the simpler measures have been tried without considerable success, and especially in those cases where the Achilles is short, a most successful anatomical and functional result is obtained by open operation to shorten the inside ligaments and the tibial tendons (lengthening the Achilles at the same time), thereby producing an artificial adduction or varus con-

tracture, and giving all the structures on the inner side of the foot time to tighten up and strengthen. The operation is done through a long curved incision behind and below the inner malleolus, extending from three or four inches above to the same distance in front of the malleolus. The Achilles is split longitudinally, lengthened by one-half or three-quarters of an inch, and sutured. The tendon of the tibialis posticus is split and divided behind the malleolus; its distal portion is pulled through the tunnel and turned forwards. Next the inferior calcaneo-navicular ligament and the adjacent part of the deltoid ligament are pleated as tightly as possible, and thoroughly shortened. The tibialis posticus tendon is restored, and shortened behind the malleolus by three-quarters of an inch. A tiny slip from the tendo Achillis is grafted into the tibialis posticus tendon behind the malleolus, giving a little of the plantar-flexion power to the adduction. All deep and superficial suturing is done with fishing gut. The foot is then put up in plaster, completely adducted and at right angles to the leg. At the end of six weeks the plaster is removed and massage and exercises begun. In another week or two walking is permitted in boots, prepared in the meantime, and fitted with wedges, arch supports, outside steel to the knee and T-strap. The appliance is worn from three to six months, re-education taking place the while, and is finally dispensed with when the feet are strong enough to maintain the new position. Short of this procedure, any case of short heel

tendon in a child should be treated by subcutaneous tenotomy before entering upon the foot drill.

A very commonly occurring disability in adults is that resulting from dropping of the anterior transverse arch formed by the metatarsal heads, and is characterized by pain and swelling of one or more of the metatarso-phalangeal joints (most commonly the second and third), tenderness on pressure, inability to take weight on the front of the foot, and by one or more callosities beneath the affected joints. Normally, in the unweighted foot, the heads of the first and fifth bones form extremities of the arch, but on weight-bearing the strain should be equally divided between the five, and all should share with the toes in the "push-off" of the foot in walking. In abnormal conditions the heads of the second and third, which normally form the top of the arch, are depressed, at times below the others, and in the lead off in walking most or the whole of the strain falls either on the balls of the second and third toes only, or, in other cases, on a triangle formed by the balls of the first and second toes and the inner side of the distal phalanx of the great toe. Almost invariably a dropped anterior transverse arch is associated with a valgus or everted deformity of the big toe (hallux valgus), and with hyperextension and "clawing" of the lesser toes. The hallux valgus may be so severe that the second toe lies above it, or beneath as a "hammer" toe. The claw deformity of the lesser toes, and their

huddled inverted position towards the dorsum, absolutely prevent their flexion function in gripping the ground; thus they are unable to assist in the "push-off" of the foot in walking, and an undue amount of weight-bearing strain falls on the too local area of one or two metatarsal heads. The cramping of the lesser toes in their dorsal position by the "toe-cap" of the shoe causes corns to appear on the more prominent inter-phalangeal joints; the latter add their quota to the painful disability. The width of the foot at the level of the metatarsal heads is distinctly increased, or, as it is said, the foot is "splayed." Even in the unweighted foot a hollow appears on the dorsum, just behind the toes, bridged by the extensor tendons, instead of the rounded fulness in the normal foot. The separation of the metatarsal bones is most marked between the first and second, and it is the inward projection of the first metatarsal head (associated, maybe, with an irritative exostosis or bunion) which exaggerates the valgus deformity of the great toe in the opposite direction. In the more acute conditions one or more of the metatarso-phalangeal joints may be very swollen and tender, and X-ray evidence shows this is very rarely infective in origin, but in most cases mechanical. The big toe joint is occasionally restricted in flexion and extension, but the typical "hallux rigidus" is quite a different clinical condition. General toe deformity and dropping of the anterior transverse arch, as here described, is almost always a "boot deformity," and due to

the use of boots or shoes the toe portions of which are too pointed and conical, of insufficient length, tapering outwards along the inner border, and too thin, narrow, and curved transversely. The deformity is probably assisted by pointed stockings. On passive movement the toes fall naturally into a position resembling that assumed in the shoe. The condition is also predisposed to by the use of heels of undue height, in which the toes, in standing, tend to be thrust forward into the narrow toe-cap of the shoe; the higher the heel, too, the less the weight on the back-foot, and the more on the already overworked one or two metatarsal heads. It follows, therefore, that higher heels have not the utility in the treatment of fore-foot conditions which they have in cases of long arch strain; if both parts of the foot are disabled we must effect a compromise. The localized nature of the fulcrum, i.e., the use of the inner one or two balls of the toes instead of the whole five in the "push-off" in walking, is the result of walking with the feet well everted, and not letting the toes swing in and point to the middle line in the final part of the step; this habit, combined with the shape of the shoe, also operates in the production of hallux valgus.

The majority of people habitually wear shoes that are too narrow across a line joining the first and fifth metatarsal heads. Up to a point just behind the joints the snugger and narrower the shoe the better, and a good transverse grip of the metatarsus is very desirable; but at the heads of the

bones the sole should be quite as wide as the foot. Otherwise, when the shoe is new, the first and fifth joints and their toes are compressed laterally, and the stiffness is experienced which is "ordinary in the new shoe." After a little use, however, the "upper" behind the toe-cap stretches transversely; but as the sole does not stretch correspondingly, the metatarso-phalangeal joints of the first and fifth toes are hung up in the upper, so to speak, and are kept at a higher level than the intermediate toes, whereas the reverse relation should obtain. This defect is increased by the custom of making most soles convex downwards from side to side, affording still less support to the central metatarsal heads. Hyper-extension of the toes produces flattening of the anterior transverse arch, and, on the other hand, nothing preserves this arch so much as a strong and complete toe flexion range and function. In pointed narrow toe portions the toes can exercise no flexion function whatever, and it is not to be wondered at, therefore, that in this unfortunate combination of adverse circumstances, fore-foot troubles are so common. The sole of a good shoe should be quite flat from side to side. From before backwards it will need to be curved, as this "rocker" action is essential for comfortable walking; it is known as the "spring," and its extent is gauged by the maker after the height of the heel has been decided. It should not be more than absolutely necessary, as it predisposes to extension of the toes. A good sole should also be as stout as possible, consistent with

the weight most comfortable for the patient. A thin sole localizes the body weight too much on the lowest metatarsal heads, but a stout one distributes the thrust of the ground more evenly over the fore-foot. It is common knowledge that a new shoe, before the sole has become soft and moulded, is much more comfortable in metatarsal weakness than later on when it has been broken in. The inner border of the toe portion need not, except in children, be dead straight to the toe tip in an antero-posterior direction; a line such as this is decidedly ugly, and a good shoemaker can give way a little in this respect in the general design of the toe portion. The outer border should be rounded sufficiently to avoid pressure on the tips and dorsal surfaces of the three outer toes. Where width is demanded, the length is usually increased to keep the proportion, but in general a lady's shoe should be two, and a gentleman's three, "sizes" longer than the foot, as measured to the tip of the big toe. A shoemaker's "size" is one-third of an inch. A well-curved waist to the shoe is of benefit in giving some support to the metatarsus, but if more support than this is required, especially in the transverse direction, either a rounded pad of felt can be applied in the shoe just behind the positions of the depressed metatarsal heads, or, what is more convenient, a metal support can be worn which snugly fits the long arch, but is also raised transversely at its front end to exercise pressure on the metatarsus and relieve the balls of the toes from undue weight-bearing strain. These anterior

transverse arch supports are usually made of light metal, but as metal may be found hard and unyielding to a very painful foot, a more comfortable support may consist of a removable leather insole furnished with a pad of sponge rubber. Some surgeons prefer the "metatarsal bar" to a support within the shoe; this is a piece of leather about half an inch thick, and an inch wide, nailed slightly obliquely across the sole immediately behind the position of the metatarsal heads. It serves to stiffen the sole, and transmit the thrust of the ground to a point just behind, instead of on, the metatarsal heads. Patients vary in the way they take to one or other method. As a more permanent support, to be used after the patient has had experience of a removable arch plate, special shoes may be made in which a support for the anterior arch is incorporated. The lasts on which the shoes are made are hollowed out immediately behind the site of the metatarsal heads, to a degree consistent with comfort and efficient support. The inner sole follows the shape of the last, and the space between this and the outer sole is filled with cork. Thus a thick rigid sole is supplied (though nothing is obvious externally), and the support of the front arch merges gradually into the waist of the shoe.

The condition can be so acute, and the fore-foot so swollen and painful, that it may be necessary to take the patient off her feet altogether for a week or two, and treat the injury as one would any other case of acute synovitis. Some cases may show very

little deformity, perhaps only some broadening of the metatarsus, but yet be subject to severe pain at times, more closely resembling the true cases of Morton's metatarsalgia. Where swelling is not prominent, and in the absence of the circumferential grip afforded by a proper shoe, the method of firmly strapping the foot transversely behind the metatarsal heads never fails to give great relief; it may be combined with an anterior arch support in the shoe, or a metatarsal bar, or the strapping may include a felt pad in the sole just behind the tender joints. In the more chronic stage, with the idea of promoting flexion function of the toes, which in itself is the greatest restorative of the transverse arch, special exercises should be given, preceded if thought desirable by tenotomy of the extensor tendons, and subsequently holding the toes down into flexion for several weeks by the use of a " half-sole plate " and tapes. Recently it has been suggested that the dorsal interossei muscles are important in bracing the metatarsus and holding its bones together, and their development by the method of faradism appears to have been attended with satisfactory results.

DEFORMITIES AND DISABILITIES OF THE FEET DUE TO PARALYSIS.

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In this our second lecture we shall be concerned with those deformities and disabilities of the feet which result from paralytic lesions of some or all of the muscles of the foot and leg below the knee-joint. We shall deal briefly with the primary causes of such paralysis; with the various types of deformity which are liable to follow, and their correction; and with the operative and other means whereby we prevent the recurrence or relapse of deformity.

Paralytic deformities are classified as congenital and acquired, the latter constituting the great majority, but for the practical purposes of treatment it will be more useful to adopt the usual classification of diseases of the motor nervous system, separating those which result from lesions of the upper motor neurons from those due to the lower motor cells, the lower neurons. This division separates the cerebral or spastic paralyses, which are characterized by stiffness of joints and

rigidity of muscles, by ataxy, inco-ordination, or loss of control of muscular movement, rather than by actual loss of power. It is true that in the cerebral paralyses there may be motor weakness, but often this is comparatively insignificant, and our problem is essentially to deal with the loss of inco-ordination. On the other hand, in the lower neuron paralyses the principal defect is a real paralysis or loss of power in one or more muscles or groups of muscles. In these we have flaccid joints and flabby muscles instead of the rigidity characteristic of the upper neuron group, and the loss of control consequent upon the paralysis is proportional to the amount of muscle paralysed, there being no real ataxy from loss of cerebral control. In the cerebral or spastic group are included hemiplegia, involving one arm and one leg; diplegia, or double hemiplegia; paraplegia, involving both legs only but not the arms; and occasionally monoplegia, confined to one limb only. The cerebral paralyses may be either congenital or acquired, and occur as the result of mal-development, intra-uterine disease, birth injuries, and meningo-encephalitis in childhood or infancy, or to the various causes occurring in later life, such as hæmorrhage, embolism, thrombosis and other diseases which, at some spot or other along the whole tract of the upper neuron, from its cortical cell to its termination in one of the enlargements of the cord, impair its tissue and destroy its function in conveying voluntary impulses and restraining the excitability of the spinal

reflexes. Thus, damage to the upper neurons limits in varied degree the voluntary control of the limbs, and in this sense there is also loss of power. The impairment of the power of inhibition of the spinal reflexes by the higher centres causes exaggeration of reflexes, and the limb muscles become spastically contracted. The so-called muscular balance is upset, and the joints are forced into certain positions, which later become fixed by adaptive changes in the tissues. In cerebral paralyses the nutrition of the muscles is not interfered with, nor is the circulation, and thus there is only little muscular wasting, due rather to want of use. For the same reason the electrical reactions of the muscles are normal. With the physical ataxy in these cases is found also, in varying degrees, some extent of mental inco-ordination or mental impairment. From the point of view of re-educative development this deficiency in the physical and psychic control is the most important feature in cerebral paralyses.

Turning to the causes of paralysis due to lesions of the lower neuron, we find that they are very numerous, and, as in the cerebral type, may be congenital or acquired. By far the commonest cause met with in orthopædic practice is anterior poliomyelitis, or infantile paralysis. In fact, the flaccid type of paralysis due to poliomyelitis and the spastic paralysis due to cerebral lesions include, to all intents and purposes, the vast majority of peripheral paralyses met with in everyday orthopædic practice, and are the types

we have chiefly in mind in the description of the methods of treatment set out in this lecture. In poliomyelitis the lower neuron suffers its damage in its nerve cell, and the other conditions leading to lower neuron paralysis have the lesions variously in the nerve cell or in some part of the nerve fibre, or at times perhaps in the muscle itself. Next in frequency to infantile paralysis is that resulting from neuritis from various causes, and, as we have seen so much in the war, from actual injury to the nerve trunks themselves. Less common paralytic conditions of the lower neuron type are those associated with spina bifida, where the nerves are damaged at their origin and lead to various types of paralytic foot deformity. Other rare conditions of paralytic foot are those associated with the Charcot-Marie-Tooth type of progressive muscular atrophy, and with hereditary ataxic paraplegia, or Friedreich's disease. As contrasted with the effect of cerebral lesions, we find that injury or disease of the lower neurons or spinal nerves is characterized by diminution instead of augmentation of the muscular tone, by slack muscles and flaccid joints rather than by muscular spasm and rigidity. Muscle wasting is much more marked, and is proportional to the number of muscles involved. Circulation changes may be obvious in the cold blue livid appearance of the skin, and in winter time by tendency to erythematous patches. The reflexes are never exaggerated and are frequently quite absent. The want of growth of the whole limb or a part of the

limb, as compared with the opposite side, may be considerable, but the want of growth in length bears no proportion whatever to the extent of the muscular paralysis. What deficiency of movement there is, is seen to be due to the absence of one or more muscles, and is not an exaggerated or inco-ordinated movement. The difference between the effect of upper and of lower neuron lesions has been expressed by saying that the upper neuron lesion causes a paralysis of movement, whereas the lower neuron gives paralysis of muscle. Although it does not really concern us now, it would, for the purposes of differential diagnosis, be advantageous to mention here the existence of paralytic deformities of the foot due to purely functional or hysterical causes. In a few of these cases no organic disease or weakness may be detectable, but in the majority there is some slight causative lesion, in itself trivial, which gives rise to a very exaggerated condition of deformity and disability. Many such cases occurring in the foot have been met with resulting from war injuries, the commonest type being the adduction or inversion deformity, the so-called "functional varus." In civil practice, however, other types of deformity from this cause are met with. In the earlier stages of a functional deformity the treatment will be purely by psychotherapeutic means, providing always that the initial slight injury has been dealt with as far as possible. But if the functional condition has persisted for any length of time there is always some

liability to organic contracture supervening, and this element of the trouble may need to be dealt with by ordinary orthopædic means before any results can be hoped for from the suggestive treatment of psycho-therapy.

Broadly speaking, the movements of the foot relative to the leg occur in four directions—up and down, and from side to side, and therefore we find the leg muscles ranged roughly into four groups, whose function it is to subserve the movements in these various directions. The arrangement of the muscles thus in two groups of mutual antagonists is sometimes referred to as the "muscle balance," a term applied generally to the arrangement of power about any joint. But if we consider the ankle and foot movements, thinking only of the action of the unweighted limb, we find that, as regards the number, bulk, and power of the muscles, the antagonistic groups by no means balance each other. The dorsiflexors of the foot at the ankle-joint are a group consisting of the tibialis anticus, extensor longus digitorum, extensor proprius hallucis, and peroneus tertius; the plantar flexors constitute a group consisting of the calf muscles, flexor longus hallucis, flexor longus digitorum, tibialis posticus, and the peroneus longus and brevis. Now the total power of the dorsiflexor group is only about one-fifth of that of the whole plantar flexor group. Similarly the adductor or inverting muscles of the foot vastly preponderate in power over the abductors or evertors. But the so-called balance

around the ankle-joint must not be considered without reference to the two functions of the foot—weight bearing and leverage action. Eversion or abduction of the foot is the standing posture favoured by gravity and is maintained largely by the weight of the body acting vertically through the ankle-joint towards the inner side of the foot. Again, dorsiflexion of the foot is done against very little resistance as compared with the load on the calf muscles inflicted by the body weight when the heel is lifted from the ground in propulsion. Thus the convenient term "muscle balance" will be a misnomer unless we are careful to remember that it applies not to the free and unresisted antagonistic movements about a joint, but to the leverage function and the load undertaken in the normal physiological action of that particular part of the body.

Having considered the arrangement of muscles in antagonistic groupings about the ankle-joint, let us now examine and find out how deformity may be produced. The two principal deforming factors must be the natural load or resistance, and the absence of power in one or more muscles to counteract the resistive effect of that load. Or, as regards the latter factor, it may not be an absence of resistance on the part of the muscle, as would be the case in a lower neuron or flaccid paralysis, but that the muscle was in a condition of exaggerated tone or spasm, and so tending to overdo its work and overweight its antagonist, as we see occurring in the spastic condition charac-

teristic of the upper neuron lesion. The exaggerated action of the calf and inner tibial muscles in a spastic leg may be so great that the weight of the body is unable to overcome the muscular contraction in these at any time in the walking step, and unable therefore to assist the dorsiflexor muscles. Thus we may get a permanent condition of deformity where the patient is unable to bring the heel to the ground, and walks on the outer border of the foot, the whole foot being in a condition of equino-varus deformity. If, then, one or more muscles taking part in a group movement are out of action from actual paralysis, or if, again, the antagonistic group is over-active on account of spasm, the natural easy condition of the joint, the position of rest or so-called "middle position," is disturbed, and the foot adopts a new habitual posture wherein the weaker group is overlengthened and the stronger group of muscles shortened. When the foot is weighted as in walking, gravity or the body weight will or will not assist the weakened group, according to which group is involved. Thus weight bearing would assist the dorsiflexors and abductors of the foot, but always works against the plantar flexors and the inner tibial muscles. Deformity at its onset can always be easily corrected by passive movement, but if its progress is not guarded against it tends gradually to increase and become more exaggerated. The overacting muscles on the concave side of the deformity become gradually shortened from their over-

activity, while those on the convex or weakened side become more and more overstretched and increasingly unable to resist their lusty antagonists. The contractures on the concave side tend to become more fixed by the adaptive shortening of all the soft tissues to the new position, so that ultimately the deformity cannot be corrected by passive movements. Even the contracted tissues will eventually show a loss of power, the want of healthy exercise leading to a disuse atrophy. Later still the altered shape and function of the foot will lead to alterations in the configuration of the bones themselves, as enunciated in the law of Wolff. While weight bearing and normal function acting on ill-balanced muscular groups are the chief factors in the production of deformity, yet there are other influences. For instance, the influence of gravity in producing a dropped and inverted foot is exercised during the time the patient is lying in bed unless care be taken to guard against this by the use of right-angled splints; so also when the foot is off the ground and unweighted, as part of the act of walking, gravity favours plantar flexion and is working against handicapped dorsiflexion muscles. It has been thought that the patients who lie for a long time in bed are liable to get dropped feet and contractures of the heel tendons merely as the result of gravity acting on the feet in the recumbent position, producing the so-called talipes decubitus. It is pretty certain, however, that dropped feet cannot occur in such cases, however

long the patient remains recumbent, unless there has been some definite neuritis leading to temporary paralysis of the dorsiflexor muscles, or some such condition as spinal caries, for example, giving rise to spasm and overaction of the calf muscles, thereby inducing plantar flexion. Whenever there is a question of the existence of any paralytic or paretic condition, whether due to upper or to lower neuron lesions, care should be taken to provide splints to keep the feet at right angles with the legs, and to remove the pressure of the bedclothes from the toes by means of a cradle. The production of contracture in a joint by mere position alone is a very uncommon occurrence, particularly in children, as is shown by the fact that joints can be fixed in a contracted position in plaster of Paris for months at a time and be found quite movable and easily corrected immediately the plaster is removed. Deformity as described above, resulting from the want of balance of the muscle groups about the joint, and due either to the overaction of one group on account of spasm or to the deficiency of action of the other group on account of actual lower neuron paralysis, is known as the " primary " deformity, in contradistinction to later results which occur as the direct effect of the initial deformity, the latter being known as " secondary " deformity. For example, in a case where the tibialis anticus alone of the group of dorsiflexors is paralysed, hyper-extension of the toes at the metatarso-phalangeal joints may result subsequently from overaction of the long

extensors of the toes in their effort to increase the dorsiflexion power. The exaggerated action of dorsiflexors tends to pull the toes backwards into extension rather than dorsiflex the foot as a whole at the ankle-joint. Again, in cases where the calf muscle (gastrocnemius and soleus) is paralysed, while the long flexors of the toes are intact, a condition of the foot is seen in which the heel drops and the os calcis becomes vertical in position instead of transverse. The dropping of the heel does not lead to exaggerated dorsiflexion of the forefoot, but the combined action of the long flexors of the toes and of gravity on the forefoot leads to an approximation of the anterior and posterior pillars of the long arch, with increasing contracture and fixation of the soft tissues of the sole. Thus at first the plantar fascia becomes markedly contracted, and later also the intrinsic muscles of the sole, and there is produced one of the most marked conditions of increase of long arch or "pes cavus" that can possibly be met with, the cavus being a deformity secondary to the primary one of the dropped heel. Another well-known instance is that of the abducted, everted, or valgus foot, a condition which may at first sight appear to be due to want of action of the tibialis posticus and anticus muscles. On closer examination, however, it is found that the primary deformity is a mild degree of equinus, due to contracture of the tendo Achillis. This primary deformity resulted from some weakness of the dorsiflexors. In the effort to produce complete dorsiflexion action in walking, the upward movement

of the foot is bound to be restricted by the short tendo Achillis, whereupon the dorsiflexion is continued not at the ankle-joint, but at the sub-astragaloid joint, which necessitates the foot being well abducted or everted in the effort to procure more dorsiflexion. In such a case close examination may show that the tibial muscles are not at all at fault, or perhaps have suffered and have become weakened as the result of the persistence of such an abducted position in walking, and so the case which was at first sight primarily an eversion or valgus deformity is found to be really an equinus or dropped foot deformity, with the valgus element quite a secondary thing. It has been pointed out in a previous lecture on mechanical disabilities of the feet how frequently this slight contracture of the heel tendon is the potent factor compelling the assumption of the abducted or flat-footed attitude in walking, and how necessary it is to test the dorsiflexion range of movement at the ankle-joint by passively dorsiflexing the foot when it is in an inverted or varus position (pp. 130-132). The severer degrees of deformity are seen in those cases showing the greatest disturbance of muscle balance, i.e., where one group is extensively paralysed while its antagonistic group is not, and this is what might be expected. On the other hand, where the paralysis is very extensive and more or less complete we do not find nearly such severe degrees of deformity, as for example in total paralysis of all the muscles acting over the ankle-joint, the so-called "flail ankle," where

the foot, although it will fall into any position according to the effect of gravity, is yet quite soft and shows no rigid contracture except possibly a slight shortening of the heel tendon; the latter fact shows that gravity itself must have some direct effect in producing contracture quite apart from the disturbance of the muscle balance. A totally flail ankle tends to fall into the position of equino-varus when it is unweighted, and usually into valgus when weight is assumed, unless the joint be so insecure that the foot turns right over into a marked varus position, the patient then assuming weight on its outer border or even on the dorsum. While discussing the production of deformity, we must mention the effect of want of growth both of the leg and of the foot. Defect of growth varies much in different cases and bears no relation whatever to the extent of the paralysis. This want of growth can be a troublesome feature when it makes the affected foot much smaller than the other, as it creates the permanent necessity to have the boot especially made. It will be seen later that the presence of a deformity by no means signifies the existence of permanent paralysis of the muscles or groups of muscles originally affected, and whose weakness leads to the production of the deformity. The nerve cells supplying such muscles may have long since recovered, and be waiting to resume their function of controlling their own muscles, while the latter are functionless as the result of overstretching or of contracture. In such a case we are dealing with *pseudo-* rather than with *actual*

paralysis, and the complete correction of a deformity may be attended with full restoration of function, without liability to relapse.

As a working rule it is important to remember that the onset and production of deformity is the result of some neglect in treatment, either on the part of the patient, the masseuse responsible for the physiotherapy, or the surgeon. This assumption is true for the great majority of cases. An effort should be made, as early as possible in the treatment of the case, to discover which muscles are affected, and precautions must be taken to prevent those paralysed from being overstretched and further weakened, and to prevent muscles showing undue spasm from contracting and overstretching their relatively weakened antagonists. In the early history of the case the paralysis of the leg and foot may be very general, when all that can be done in the matter of preventing deformity by splintage is to keep the foot in the position necessary for correct function, i.e., correct walking and standing, favouring the recovery of those muscles which, when the leg is used again, will be subject to the greatest weight-bearing strain. Thus, with a foot absolutely paralysed, when the patient is yet recumbent, we should by means of splints hold the foot at right angles to the leg, to favour the recovery of the dorsiflexors and prevent the tendency to foot drop which must otherwise inevitably occur. Knowing also how important it is to preserve the strength of the inner tibial muscles, we should not only keep the foot

at right angles but also adduct or invert it slightly. Later the splint arrangements can be modified to favour the affected muscles by fixing the foot in such a position that these are not pulled upon and overstretched. At the same time the application of electrical and exercise treatment can be more specifically determined. In the later stages, when the patient is getting about again, we must remember that unless the muscle balance is absolutely restored by the full recovery of the muscles we shall inevitably have deformity slowly occurring if precautions be not taken. The same applies, of course, after a deformity has been completely corrected by surgical and other means. The notorious tendency to relapse which exists in these cases is the result of the persistent ill-balance, together with the effects of gravity and weight-bearing. The instrumental and operative measures necessary for the prevention of deformity or of its relapse we shall discuss later on. But in spite of every care on the part of all concerned in the treatment, recurrence of deformity will continue to take place in many cases. The progress is often so slow that until well marked it is undetected by the patient, who therefore does not seek surgical aid until the condition is gross. Boots and appliances will wear out and become less effective with time, and it must happen that in some cases we shall need to repeat the corrective measures. For example, a permanent tibialis anticus paralysis is bound to lead in time to contracture of the tendo Achillis, and it may be necessary to repeat at

intervals of a few years its lengthening by tenotomy. Again, it is not an unknown thing in cases of pes cavus to have to divide the plantar fascia and stretch the arch on more than one occasion. Bearing in mind the inefficacy of boots and appliances alone to prevent the occurrence or relapse of deformity, however carefully designed, and remembering, too, that massage, physiotherapy and physical re-education is an expensive treatment which cannot be indulged in for ever, it should always be the effort of the surgeon to obviate these measures as much as possible by the more permanent surgical procedures discussed below, such as tendon transplantation, tendon fixation, and arthrodesis. In the treatment of deformities generally the importance attached to any one of the three main lines of treatment is liable to vary with the person in charge of the case. As regards feet there is not quite the same difference of opinion as formerly existed in the treatment of scoliosis, when there were rival schools, one of which treated every curve with gymnastics alone, and the other with nothing except spinal appliances, but there does occur some conflict of opinion as to the relative importance of the procedures—appliances, physical exercises, and operative measures. The instrument maker will fit any foot, however misshapen, with some sort of appliance intended to assist the patient in getting about. On the other hand, the enthusiastic physiotherapist will often expect far too much from his exercise measures, and will insist on this

expensive line of treatment only, to the exclusion of adventitious support and surgical assistance, forgetting what a great tax on a weak and disabled foot is afforded by the sheer effect of weight-bearing. It remains for the surgeon to apportion the proper values of each of these lines of treatment in any particular case, and to appreciate the importance of them all under the various conditions that may occur. Orthopædic surgery has in the past been much too limited in its actual surgical procedures, and has often been content with such simple things as subcutaneous division of a tendon here and there, followed up by a wrenching of the foot into some sort of correct position, the so-called "forcible rectification," done blindly with the aid of wrenches and other barbarous machines. This kind of conservative orthopædic surgery is crude and not very precise, and often does not go far enough. The object is often only the immediate correction of the deformity and the subsequent inevitable use of some boot and appliance, which more often than not does not prevent its recurrence. With modern surgical technique much more than this can be done in correcting deformity, by open surgical operation to restore muscle balance and prevent relapse of deformity by rearrangement of tendons and by joint fixation. There is no doubt, however, as to the importance of long-continued physiotherapy, as shown by the length of time during which these seemingly paralysed muscles will continue to increase in strength, and by the

appearances of the muscles at open operation which will be described later on.

In lower motor neuron or spinal nerve lesions, typically seen in infantile paralysis, there may exist any degree of paralysis, from the involvement of a part of or a single muscle to complete absence of function in every muscle in the limb, but certain degrees of partial paralysis, with corresponding deformities, occur fairly frequently. The number of cases of complete permanent paralysis of the ankle and foot, the so-called "flail foot," is proportionately not very great, as the majority of cases eventually get some restoration of power in one or more muscles or groups, although at times, as for example in the sole of the foot, it is not of very much value. We are now considering cases as they appear about six months after the onset of the disease, when they have passed the stage in which progress towards recovery is rapid, and have entered upon the final stage, which may exist for years, when improvement may even yet steadily though slowly occur with efficient treatment. In the first month or two of the illness the degree of paralysis is at its worst, but thenceforward for a few months, in most cases, a considerable degree of improvement is attained. In infantile paralysis the commonest type of deformity resulting from partial paralysis is that where the tibialis anticus muscle alone is paralysed. The persistence of the paralysis in this muscle is so frequently met with that one cannot help wondering why all the other muscles

have got well except this, a fact which would suggest that it must be due to some mechanical factor acting during walking and getting about, rather than to the permanent destruction of its particular controlling nerve cells in the spinal cord. Deficiency of this muscle, which supplies more than three-fifths of the power of the dorsiflexion group, leads to a very characteristic deformity of the unweighted foot, typical of an almost pure overaction of the peroneus longus. The foot is slightly abducted, and rotated so that the inner border is low and the outer border high, with some depression or plantar flexion along its great toe portion. When walking and standing the foot is somewhat abducted and everted, but the lifting of the outer border is not so much. The foot is therefore everted rather than abducted or valgus, the latter position being more characteristic of paralysis of both tibialis posticus and anticus muscles. The absence of the dorsiflexion power afforded by the tibialis anticus throws the balance distinctly in favour of the calf muscles, and in this deformity there is invariably a contracture of the tendo Achillis, which, although corrected, will inevitably recur after a few months unless the balance is restored by adding the peroneus longus to the dorsiflexion power by grafting. The persistence of an overlooked contracture of the tendo Achillis will lead to continuance of the eversion posture in walking, which scarcely helps the restoration of the tibialis anticus muscle, and it frequently happens that the mere lengthening of

the tendo Achillis by tenotomy and the retention of the foot in an inverted position for some time, assisted by walking function in inversion, leads to the restoration of an otherwise seemingly permanently paralysed tibialis anticus. No transplantation should therefore be done until the heel tendon has been lengthened and function established for some time in the inverted position of the foot. The deformity resulting from the paralysis of the tibialis anticus muscle is, therefore, a condition of slight equinus with eversion, or *equino-valgus*. The next commonest type of deformity in infantile paralysis is that due to the complete loss of all the muscles supplied by the external popliteal nerve, the peronei and the dorsiflexors, giving a dropped and inverted foot, or *equino-varus*. The plantar flexors and adductor muscles may be spared, but as a rule they show some deficiency of power, probably largely conduced to by the absence of sufficient antagonistic stimulation. A very disabling deformity is that due to paralysis of the calf muscles, the gastrocnemius and soleus, and is characterized by a dropping or vertical position of the heel instead of the transverse normal shape. The calf muscle is three times as strong as all the other muscles of the leg and foot put together, and even though, as is frequently the case, the long flexors of the toes and the tibialis posticus be spared, the latter can in no way assist the impaired function of raising the body on the toes. Dropped heel deformity illustrates extremely well the adduction

or inversion function of the calf muscle which occurs at the time of the plantar flexion, as a paralysed calf muscle causes not only dropping of the heel, or *talipes calcaneus,* but also abduction of the heel, or valgus, and thus is always produced the combined deformity of *calcaneo-valgus*. The calf, like the inner tibial muscles, are anti-gravitational, and when they are paralysed, gravity, acting through the body weight, gives them no chance whatever. As mentioned above, in calcaneo-valgus there is nearly always a secondary deformity, *cavus,* from approximation of the anterior and posterior pillars of the long arch, with contracture of the plantar fascia and other tissues of the sole. No cavus is ever so marked as that which occurs with a paralysed heel. The production of cavus and contracture of the sole is due to the action of the long flexor muscles of the toes, as shown by the fact that if these tendons are transplanted to augment the heel-lifting power the cavus gradually disappears when weight bearing is again assumed, and without the necessity for division of any of the plantar structures. Another deformity, *valgus,* when not due to paralysis of the calf muscle, may result from paralysis of both the tibialis anticus and posticus muscles together. Although the tibialis anticus is the commonest muscle permanently paralysed, the tibialis posticus is one of the rarest. Deformity due to combined tibial paralysis is not so common as those mentioned above, and it leads only to abduction of the foot, or valgus, and not, as a

rule to flattening of the arch or *planus*. Real paralytic planus is much more frequently seen in some cases of spastic paralysis, although it is not a common deformity even in these. Pure paralytic *varus*, due to the absence of the peronei only, is the rarest deformity met with in infantile paralysis, as the peronei are rarely if ever involved without one or more of the dorsiflexor muscles. Of moderate frequency is total paralysis of all the muscles around the ankle-joint, giving rise to a flail foot, which assumes a general equino-varus position, with the toes tending to flexion in all their joints. Acquired increase of the long arch, or *pes cavus*, as a primary deformity is not so typically the result of infantile paralysis, although sometimes it certainly can be due to this disease. As a rule it is bilateral, although usually more marked in one foot. Frequently there is no indication as to its cause, and it is possible that some cases are due to an overlooked neuritis occurring in an infectious fever, such as scarlet fever, while others may result from some disease of the nervous system. Pes cavus is almost always associated with a more or less degree of *clawed toes*, or toes which are hyper-extended at the metatarso-phalangeal and flexed at the inter-phalangeal joints. The proportional severity of the arch and toe contractures varies in different cases; in some the contracted arch is much more marked, while in others it is the contracture of the toes which leads to the loss of function and painful disability. As a rule cavus is associated

with slight varus or inversion of the foot, either due to overaction or spasm of the tibial muscles, or to some want of strength in the peronei. It is frequently insisted that pes cavus is associated in many cases with a contracture of the tendo Achillis, but this is the exception rather than the rule. The point is important, as it is vital not to tenotomize the Achilles unnecessarily; a too long heel tendon is very disabling. The resemblance of the clawed toes to the position assumed by the fingers in paralysis of the median and ulnar nerves has led some to think that clawed toe deformity is the result of paralysis of the interossei and lumbrical muscles of the foot, but it is quite certain that paralysis of these intrinsic muscles does not exist in many cases.

In lower neuron or spinal paralyses due to other causes than *infantile paralysis*, the types of deformity met with are much the same as those described above as occurring in anterior poliomyelitis. A *neuritis* which is very peripheral may lead to paralysis only of the intrinsic muscles of the sole of the foot, with the production of clawed toe deformity, and some increase or hollowing of the sole due both to the impaired toe flexion function and to the want of tissue bulk resulting from the muscular atrophy. In neuritis of the larger branches and nerve trunks, and also in cases of actual nerve destruction, as from *gunshot wound*, the kind of deformity occurring will depend on which of the two popliteal nerves is damaged. Injury to the external popliteal nerve will cause an

equino-varus on which later some cavus supervenes. The peroneal and dorsiflexion muscles are inactive, and the foot drops both at the ankle and mid-tarsal joints. Contractures of the tendo Achillis and of the long flexors of the toes occur from adaptive shortening. On walking the foot will be turned in, with weight thrown on the outer border, and with tendency to adduction and flexion of the toes, so that pressure comes on their tips and nails. If the anterior tibial nerve is affected below the origin of the peroneal supply, dropped foot occurs and contracture of the heel tendon, but walking is done with the foot everted and valgus. A lesion of the internal popliteal or posterior tibial nerves does not give such an obvious deformity as that resulting from the external popliteal, as in the unweighted foot gravity tends to hide the deformity. In walking all the weight will come on the heel, there being no toe and heel action, and the foot will become abducted or valgus, with prominence of the inner ankle. In paralysis of the plantar flexor muscles, particularly in adults, the deformity is not nearly so important as the disability. In children, however, in process of time we get the dropped heel and dropped forefoot condition, with marked increase of arch and valgus deformity, as described above. Of the rarer conditions, *spina bifida* may cause any of the usual deformities, the commonest being paralysis of plantar flexion, with calcaneo-valgus deformity; but in other cases we may have varus or equino-varus or a complete flail foot. The

Charcot-Marie-Tooth type of *muscular atrophy* gives an equino-varus deformity, on which supervenes cavus and clawed toes. Here the peroneal muscles are principally affected, and also the dorsiflexors. The progress of the atrophy is so slow, and the inner tibial muscles involved so late, that it is well worth while transplanting the tibialis anticus tendon to the outer border of the foot, where it will act as an evertor as well as a dorsiflexor. The deformity seen in *Friedreich's* disease, or hereditary ataxic paralegia, is very similar to that occurring in Tooth's muscular atrophy, a combination of equinus at the ankle-joint and increase of arch by further dropping of the forefoot at the transverse medio-tarsal joint, and clawed toes. We have yet to mention the type of foot characteristic of the lesions of the upper motor neuron or *cerebral paralyses*. As compared with those occurring in the spinal paralyses, the deformities resulting from spastic conditions are not nearly so extreme. This is what we should expect, as in spastic paralyses we are dealing with ataxy or inco-ordination of group movements, whereas in spinal or flaccid paralyses we may have any combination of muscular deficiency. Another difference is that the deformity resulting from cerebral paralysis is often most marked when the patient attempts walking, and may not be so obvious in the unweighted foot. The simplest and most common condition is spasm of the calf muscle, leading to a dropped foot or slight equinus condition. Without treatment this

may in course of years become exaggerated, and a very extreme condition caused, wherein the foot is dropped both at the ankle and the mid-tarsal joints, and the patient unable to bring the heel to the ground, walking only on the balls of the toes. But in the earlier cases in children, on passive dorsiflexion being maintained for a few moments it may be found that the deformity is not fixed, i.e., is not due to an adaptive shortening of the heel tendon, but merely the result of spasm and resistance in the calf muscle, which gradually gives way before the increasing pressure of the passive dorsiflexion. Such slight degrees of equinus or toe drop are met with in the slighter cases of hemiplegia, monoplegia, and diplegia. In well-marked cases of hemiplegia and diplegia, especially those untreated and neglected, the foot may be distinctly varus as well as equinus, with toes turned in and ankle thrust out, the patient taking all the weight on the fore part of the outer border of the foot, and on the balls of the outer three or four toes. As in other cases of equino-varus deformity, when longstanding, it is liable to be associated with cavus and clawed toes. The usual mode of progression in a case of cerebral paralysis is with the hip flexed and adducted, the thigh rotated in, the knee flexed, and the foot plantar flexed and adducted. Although equino-varus is by far the commonest deformity met with in spastic paralysis, yet now and again occurs a most extreme condition of plano-valgus, or abduction of the foot associated with a complete

loss of the long arch. The foot appears to be broken across or dislocated at the transverse mediotarsal joint, with its inner border resting completely on the ground and convex in outline. Such cases are of the most extreme degree of paralytic flat foot that can be met with. It is very probable that the valgus and planus is secondary to equinus resulting from a shortened heel, and occurs quite early in life, and that it is analogous to the condition of weak ankle and flat foot resulting from a short heel tendon described in the previous lecture (p. 131). The correction of a spastic equinus by tenotomy of the tendo Achillis must be made with due care to see that the correction is not overdone, as tenotomy is liable to result in an overlengthened weak tendon, with considerable permanent loss of power in the calf muscle. The result of this is that, although the patient can now bring the heel to the ground, weight-bearing and walking are performed almost wholly by the heel, with no toe and heel action, and there is a liability to the production later of everted, valgus, or flat foot. The want of co-ordination in spastic cases renders them less satisfactory for correction of deformity and prevention of relapse by tendon transplantations than spinal paralyses. Although by tendon transplantation we may hold the foot in a better condition, yet the want of individuality of muscle action does not give us a satisfactory functional result.

We must now consider what difficulties and defects of function result from the various types

of deformity described. Disability considered from the purely physical point of view, without regarding for the moment the importance of the psychic side of the organism, depends on the two mechanical factors—*force* and *movement,* or the power of the muscles to move the limb levers about their fulcrums, and the integrity of the joints themselves, which constitute the fulcrums, to permit the leverage action. In the majority of surgical conditions, such as fractures, arthritis, etc., the prime defect is some limitation of movement in the joint, and the deficiency of power is a secondary result depending upon disuse and reflex atrophy. But in paralytic conditions, more particularly in those due to spinal nerve lesions, there is no fault to find with the joint, and the principal defect is the inability to supply the requisite and correct forces to the otherwise intact levers. In the spastic paralysis, however, we can scarcely say that we are dealing with a primary mechanical defect, as in these cases the disability is of the psychic order, and due to the want of driving force and to that inability to promote correct muscular action which results from the defect in the volitional mechanism. The essential element of the disability, therefore, in spinal nerve lesions is the loss of power and control, and the sense of insecurity caused by the loss of stability in the ankle and foot. When we consider the great weight the foot has to sustain in supporting the body, and the big load that is thrown on its mechanism in walking, we realize how important is this loss of stability. In the

upper limb we require more particularly finer movements only, making no demand comparable to the enormous effort necessitated by weight-bearing and progression. Newton's third law says that to every action there is a reaction, or in other words, there must be some "push off" point for every force exerted. For example, if we wish to exert some force or do some work with the hand and forearm, there must be some fixation point in the body from which, so to speak, that force leads or "takes off," and in arm movements this fixation point, or point of reaction, is provided by the weight of the body acting through the back when lying, through the buttocks when sitting, and through the feet when standing. To do such a peripheral action of work all the joints between the application point of the force exerted and the fixation spot afforded by the body weight must be "locked" or fixed by muscular action, by means of the so-called "fixation muscles." Nothing more than the very simplest muscular effort can be made without a tightening up or fixation of the joint muscles throughout the body. If we make a heavy thrust in the attempt to do work, we "take off" from the ground, which is our fixation point, and the ground exerts that "reaction" to our action which is enunciated by Newton. Hence we see that, if one link in this chain of joint fixation is weakened on account of paralysis of a group of muscles, work cannot be done, as there is no means of carrying the thrust back to earth. This, then, is the mechanics of

the disability of a flail joint, and the obvious mechanical solution is to stiffen that joint in some way, either by an external rigid apparatus or by a surgical operation to obliterate the joint. A joint which is uncontrolled by muscles is a source of weakness, and the patient is better without it at the moment he is attempting to do the particular work. When standing on the leg there is a continual force exerted on the foot, due to body weight, which is transmitted through the leg to the ground, the reaction of the ground forming the counter thrust. If the body is not to collapse or fall down, there must be some means of making a rigid whole of the skeleton between the centre of gravity of the body, from which the weight may be supposed to act, and the point of reaction on the ground; it is the inability to accomplish this in paralytic conditions of the foot and ankle which is the mechanical explanation of the resulting insecurity. Of first importance, therefore, in paralytic conditions of the leg and foot is the necessity for transmitting the body weight securely. But besides the elementary function of posture and weight bearing, there is also that of locomotion, where, by requisite muscular action, we are able to thrust the body weight into such a position that it may be utilized in assisting progression. If from paralysis of the calf muscles and inability to plantarflex, the body weight cannot be lifted, progression movement has increasingly to be carried out by the larger muscles about the hip-joint and of the trunk. The absence of a proper toe

and heel action demands, therefore, compensatory movement of a more exaggerated kind in the hips and trunk, a means of progression which, although effective, is not so economical or graceful; also, the strain of weight-bearing is not distributed in normal proportion on the heel and toe portions of the foot, the one or other being subject to undue strain, according to the group of muscles paralysed. The loss of security, the exaggerated compensatory muscular effort, and the undue strain on the partly paralysed group, cause general fatigue and lowered mechanical efficiency in walking and work production. Except in the early stages of some cases of neuritis, pain is not a feature tending to diminish efficiency, but it may occur later as the result of strain and deformity through function in improper position, or from the tedious secondary deformities which may develop. Thus, for example, toes will become deformed, with callosities developing on them and on the balls of the toes; and bursæ and exostoses may occur on the outer border of the foot, on the top of the instep, and on the great-toe joint. In infantile paralysis we are not usually troubled with trophic conditions, except that in winter time erythema induratum may occur on the back of the lower part of the calf, but in other conditions there is greater liability to the formation of pressure sores, or, for example, in sciatic injuries, and in cavus and other deformities resulting from spina bifida. Usually these perforating sores or trophic ulcers result directly from the undue pressure of

boots and appliances, and from neglected corns and bursæ. In equinus or equino-varus deformity there occurs an excessive flexion action of the hip and knee-joints in lifting the foot far enough from the ground to clear the toes in walking, a function which should normally be performed by the dorsiflexors of the foot; otherwise the patient would trip over his own toe-tip. If the peroneal eversion action is absent the foot may be brought down on its outer border and the ankle tumble outwards, a plantigrade position being impossible. In such cases, also, the flexion deformity of the toes is a great trouble, as the toes tend to bend underneath when the boot is put on. When there is much contracture of the tendo Achillis it may be quite impossible to bring the heel to the ground, especially if the leg is deficient in growth and the equinus deformity is utilized to compensate this diminution in length. In severe equinus all the weight may be taken on the balls of the toes, which develop callosities from undue pressure, and there may also be a secondary cavus or contraction of the arch of the foot, adding a dropped forefoot to that occurring at the ankle-joint. The tip-toe walk may in consequence be very disabling, and even if the extensors of the toes are not affected the function of the toes is impaired, both on account of the over-extension due to the effort to try to keep the foot dorsiflexed, and to the relative shortening of the long flexor tendons consequent upon the dropped foot. Calcaneus or dropped heel deformity, due to paralysis of the

calf muscles, is one of the most disabling conditions, because the weight of the body is exerted wholly through the heel in all positions of the step, the forefoot having no function whatever in taking any of the weight; thus the step is quite inelastic, and the thrust of the heel on the ground causes considerable jarring. If, as is usually the case, abduction or valgus deformity is also associated with the calcaneus, the foot is displaced laterally from underneath the ankle-joint, and the body weight transmitted to the inner side of the heel and foot. The foot is quite useless, as well as being a source of disability, and rather than this a Syme's amputation would often be preferable. Varus deformity has been discussed above with equinus; it causes cramping of the outer toes, and callosity or bursa formation on the base of the fifth metatarsal. Its chief defect is its liability to cause turning over of the foot at the sub-astragaloid joint. Valgus deformity, or abducted foot, may result from loss of the tibialis anticus alone, or of this muscle together with the tibialis posticus; or it may occur in association with a paralysed calf. It causes turning out or eversion of the foot, a position in which plantar flexion action cannot occur, as plantar flexion is always associated with inversion movement. Consequently valgus deformity must lead first to a diminution and then to complete loss of the toe and heel action, and the foot is used in walking in the position intended only for standing. A valgus foot escapes from the line of weight, which now falls to the inner

side of the os calcis, and consequently strain is thrown on the inner ligaments of the foot and ankle-joint, and on the inner tibial muscles. This may give fatigue and pain from stretching of the soft tissues, and planus, or flattening of the arch, supervenes as a secondary result. The abducted os calcis becomes impacted against the tip of the external malleolus, with the production at this spot of a localized periostitis, giving rise to pain and tenderness. The eversion of the valgus foot may subsequently cause a valgus deformity of the big toe, with loss of its function, and the predisposition to similar changes in the smaller toes. Cavus deformity, or increase of the long arch, diminishes the normal elasticity of the foot, and the tarsal joints become rigid; consequently there is not in weight bearing that normal sliding in the tarsal joints which increases the bearing surface of the sole of the foot, and which is sometimes referred to as the alteration of the arches under the pressure of weight. A too hard and unyielding foot, as in cavus, is a far greater disability than one too soft as seen in some mobile cases of flat foot. If no shortening of the tendo Achillis be associated with the cavus, the whole of the body weight is taken on the heel behind and the balls of the toes in front, a surface much too small for weight bearing compared with that of the normal foot. As cavus is nearly always associated with hyper-extended or clawed toes, the weight-bearing area is even further restricted. Hyper-extended toes have no flexion function, and in the toe and heel

action of normal walking they do not relieve the balls of the toes from undue pressure as they should. All the thrust in the "push off" falls therefore on a few metatarsal heads, instead of on all the toes and all the metatarsal heads, and hard callus formations develop, the reaction of the skin to the pressure; these corns increase the painful disability. When an equinus element exists as well as the cavus, the heel takes no share in weight bearing, which is then wholly on the metatarsal heads. Cavus and clawed toe deformity cause much general fatigue in walking, which is what we would expect from the want of suppleness in the feet, and from the impairment of the dorsi-flexion range in the mid-tarsal joint. Cavus deformity may be associated with valgus or varus, but usually with the latter. Clawed and hammer toes are a trouble, not only because they cannot flex and are therefore of no use, but also because of their liability to injury and corns. The disability due to actual shortening of the foot and leg depends on its extent, and on the possibility of compensation by the use of a thickened sole to the boot. If uncompensated, the short leg takes too much of the body weight, and the disability and weakness resulting from postural scoliosis will be added. The leg should be restored to its original length, in the slighter cases by the addition of a pad underneath the heel, which, however, throws the foot into equinus, thus limiting the strength of the toe and heel function. When the shortening is extreme, i.e., more than an inch, the raised

sole must be carried forward to the toes, or a more severe equinus position adopted by using some appliance such as the O'Connor boot, both of which prevent the toe and heel action and permit the leg to be used only as a stump or pedestal in walking. The attitude assumed in these various deformities is well shown by the position and extent of the wear and tear of the boots. No evidence of existing weakness is more satisfactory than that afforded by an examination of the boot. The equinus foot kicks out the tip of the toe, while the calcaneus foot wears away the back of the heel, no matter how high it be raised. The weakened tibials and the valgus posture are revealed by the wearing down of the heel and sole along the inner border and the falling of the "upper" of the boot over to the inner side, and the opposite result is seen in the varus foot. The valgus or flat foot also breaks down the waist and inner side of the shoe, while the cavus foot with its claw toes contracts the boot from front to back, shows the imprint of the toes on the toe-cap, and produces transverse creases just behind.

On seeing a case of foot deformity for the first time, certain investigations will be required before one can determine the extent of paralysis actually existing, the results of use in the improper position, and how much of the deformity is primary or secondary. An endeavour should be made to discover how long the paralysis has existed, and to estimate the care, as regards appliances, muscular re-education, etc., which has already been

given to the case. Our object will be to correct the deformity, as this is the only means of reducing the disability and of giving the best prospect for partial or complete restoration of function. We shall remember the two possibilities concerning the muscles which are seemingly quite paralysed, firstly, that the paralysis is *complete* and function irrecoverable on account of absolute destruction of the nerve cells or fibres; and, secondly, that the muscles may be only in a condition of *pseudo-paralysis,* and functionless because they have never been helped in any way to recover from their original knock-out blow. This pseudo-paralysis may result from overstretching or overlengthening of muscles at a time when they themselves were unable to prevent it; or, in the case of the antagonists, they may be so contracted as to be completely wasted and powerless. We shall estimate the degree of real or pseudo-paralysis according to the care that has already been bestowed. In cases of obvious neglect corrective measures must be of the simplest kind, and we shall refrain from drastic structural alterations, such as tendon rearrangements and arthrodesis, until we know the contracted or overlengthened weakened muscles have had their complete chance of repair by assisted function in the normal position, and by physiotherapy and re-education. A case of well-marked deformity may be such a combination of primary and secondary deformity, of contractures and overstretchings, and of true and false paralysis, that it may be very difficult at first to decide

what surgical interference is required. Personally, I do not consider that electrical testing is of much service in determining which muscles are and which are not paralysed, and I am inclined to attach far more importance to a careful naked-eye observation of the efforts at voluntary contraction, and, in open operative work, to the appearance of the muscle bellies themselves. In many cases of infantile paralysis it is impossible to get a response to faradism, even when voluntary contraction is apparent clinically. Positive evidence is therefore more easily obtained by clinical observation than from electrical tests, and in the latter negative evidence is of no value. In cases where tendon transplantation is under consideration, before acting purely on clinical evidence, I very often prefer directly to examine the muscles concerned through a special incision, sometimes using also the faradic current on the exposed muscles, and I determine only after this inspection the possibilities of the transplantation. The naked-eye appearances of the affected muscle vary considerably, from the healthy dark-red colour of the normal muscle to the pale-yellow appearance of a muscle that appears absolutely dead. Between these two extremes is found every variety of appearance. Sometimes the whole muscle is paler than usual, suggesting that, although somewhat degenerated, it might be able to be worked up into a better functionable condition; in others strips of obviously degenerated muscle intermingle with quite healthy fibres. It is possible that in

the latter case certain nerve fibres to the muscle have been destroyed while others are quite healthy, and that the groups of healthy muscle fibres might be developed by a process of re-education. In deformities resulting from spastic or cerebral paralyses one must not forget the condition of muscular spasm, as contrasted with that of actual shortening or contracture, which must be excluded by sustained passive stretching before the degree of contracture can be estimated. Another important thing is not only to look at the foot, but also to examine the patient as a whole, estimating the proportionate value of the foot disability, and its effect upon the whole organism. For example, equinus deformity may be secondary to a flexion contracture of the knee; an everted or valgus foot may result from some paralysis in the hip muscles; again, the deformity of the foot may be maintained by the general shortening of the whole leg. The value of any operation on the foot itself, such as tendon transplantation or arthrodesis, may be determined wholly by the strength of the limb above, and no treatment will be carried out for the benefit of the foot alone without due regard to the body mechanism as a whole. Finally, the investigation will not be complete without examining the boot for evidence of functional defect, and without seeing the patient walk and stand both in his usual boots, appliances and supports, and also bare-legged.

Before discussing details of treatment, it will be useful to enunciate those general principles

which guide us in all stages of the treatment of paralytic defects. If we see a case within a few weeks of the onset, presuming it is one like poliomyelitis which begins acutely, we first apply the principle of *rest* by keeping the patient recumbent, knowing this is essential to cure the infection, allay the inflammation, and promote tissue repair. Any acute case such as acute poliomyelitis and neuritis will be rested in bed and splinted to keep the spine and the limbs quiet for an interval varying from a few weeks to three months. As a rule in anterior poliomyelitis it is desirable to keep the patient recumbent for this time, after which the treatment is diametrically altered, and, instead of *recumbency,* we rely on *ambulation* as our main line. In the earliest stages of these diseases of acute onset the extent of paralysis is at its maximum, and it may be difficult or impossible then to determine the prognosis as to which muscles will recover and which remain permanently affected. Much pain may exist in addition to the muscle weakness, and for these reasons we adopt the splint treatment—to rest the spinal column, nerves and muscles, to relieve the pain, and to prevent the limbs assuming a fixed position incompatible with a correct standing and walking posture. While the patient is yet recumbent, and after the pain has ceased, we begin our earliest attempts at physiotherapy. Light gentle massage and electrical treatment will assist the circulation of the limbs and tone up those muscles which show diminished or no voluntary power. The

limbs must be kept warm and the gentlest active movements commenced as soon as possible. Active movements in the early stages must be *assisted* by relieving the patient from the dead weight of the limb; unassisted movements in this early stage put too much strain upon the disabled and weakened muscles, and would be distinctly harmful. For this reason it might be preferable to give graduated faradic stimulation to those muscles which will respond, stimulating them to a degree insufficient to move the limb but enough to elicit a muscular response. For the recumbent period the lightest possible splints should be used, and those positions adopted which keep the spine straight, the hips extended and slightly abducted, the knees extended, and the feet at right-angles with the legs. When it may be assumed that no exacerbation of the inflammation or no injurious stretching of muscles will occur as the result of the undue activity necessitated by standing, ambulatory treatment is begun. Nothing can stimulate the restoration of function so much as attempts to perform that function under conditions of correct position, and with requisite support to the weakened joints and careful prevention of any undue fatigue. In the early attempts at walking mechanical appliances may be necessary to maintain the patient in the upright attitude, the position in which he can endeavour to help himself. The patient must learn to stand before he can walk, but if his quadriceps muscles are so paralysed that he cannot maintain his knees extended, then, to

permit him to stand, the knees must be temporarily supported by some light splints. This principle of mechanical support is known as "splinting the weakest joint," and it may happen that more than one or two joints will require such temporary support. Mechanical appliances may also be of the nature of standing and walking machines and crutches, intended to assist the deficiency of power in the legs by the temporary use of that of the arms in maintaining balance in an erect position, and in assisting progression. Again, appliances and splints may be used in other cases not to fix the joint absolutely, but to permit slight movements only, thus assisting and protecting the muscles by relieving them of undue strain while permitting them to do a little work for themselves. Later on it will be shown that another type of appliance is that designed for correcting deformity, but this is often an expensive, cumbersome and inefficacious machine whose function is better served by the surgical procedures we have at our control. Ambulation must always be the main principle of treatment, provided that arrangements are made for the attempts at function to be done in the proper position, but we shall always take care to avoid overdoing it and thus causing fatigue, to which these affected muscles are so liable in the early stage of the disease. In the ambulatory stage also everything will be done that physiotherapy can supply—electrical treatment, massage, and, above all, active exercises. Passive movements have their use only when it

is desired either to correct some deformity or to prevent its relapse. Care must be taken to keep the affected limbs warm, as they suffer from defects of circulation and growth as well as power. Poliomyelitis cases begin to recover their power after about a month from the onset of the disease; for the next six months the rate of recovery is far more rapid than subsequently, and in the first year most of what recovery is possible takes place. But under the general good conditions afforded by walking and getting about in suitable supporting appliances, with care to prevent possible deformities, and with every facility for physiotherapy and muscular re-education, the improvement may continue slowly though steadily for many years. It is always difficult to say when recovery is complete—probably it never is complete under the best conditions of treatment—but there comes a time when, as a practical question, little more recovery is to be expected, except at a very slow rate and by very expensive physiotherapeutic treatment, and it becomes necessary, therefore, to decide what further can be done by operative means to rearrange that power which is left to the best advantage of the patient, to reduce the amount of appliance worn, and to prevent the onset or recurrence of deformity inevitably resulting from partial restoration of muscle power. The time at which such surgical procedure should be carried out will depend on many things, such as the patient's age, economic position, occupation, etc. In children there is no hurry, as their

reparative powers are more marked, and they may be kept in their appliances for years before any surgical operation is undertaken. In adults, however, it may be necessary or advisable to adopt operative treatment somewhat sooner. As a working rule, it may be laid down that no considerable recovery of power can be expected after about three years, and at this stage tendon rearrangement and joint fixation should be carried out. In cases which are chronic from the first, depending upon an irrecoverable lesion of the spinal nerves, there is no need to defer operative procedures. When dealing with a case of existing set deformity, it is maintained that the deformity should be over-corrected, by which is meant that the limb segment is carried into and maintained in the opposite extremity of movement in the joint at which the deformity has occurred. The over-correction is kept up for a period of one to two months, in some fixation splint, to overstretch thoroughly the contracted tissues on the concave side of the deformity, and to allow those overstretched on the convex side to shorten adaptively. After the fixation period, function is permitted, assisted by some appliance which prevents the deformity from recurring, but allows the utilization of physiotherapeutic and re-educative measures. Sometimes the patient is permitted to walk on the splinted foot even during the period of correction, as in a " walking plaster," in order that the function may assist the correction. Subsequent to the over-correction of the deformity the main consideration is the prevention of relapse,

which is always possible as long as there is any deficiency of power in one of the antagonistic groups. No trouble will be spared to restore the overlengthened muscles to their original strength; the cure cannot be complete nor the danger of relapse over until, with these muscles, the patient can voluntarily correct his own deformity. This is the principle of prevention of relapse by the development of the patient's own voluntary control. Should it happen that the overstretched muscles are out of action altogether and their recovery impossible, then the deficiency of the muscular power must be supplied or compensated for by some permanent appliance, or by tendon transplantation, or by tendon and joint fixation.

Turning now to the details of methods used for the correction of already developed deformity and the prevention of recurrence, we shall begin with a summary of the means at our command. It has already been emphasized that all care should be taken in the early stage of the disease to prevent deformity. The milder degrees, for example slight equinus, cavus, etc., may be corrected by massage and exercises alone, providing it is regularly carried out, but in most cases a corrective night splint will be required as well, to maintain the good position during the hours of sleep. Further degrees may require a simple operation, such as tenotomy of the tendo Achillis or subcutaneous division of the plantar fascia, followed immediately by massage, exercises, and functional use. Still more marked con-

ditions may, after the tenotomy, require forcible manipulation or correction while under the anæsthetic, and the subsequent fixation of the foot and leg either in splints or in plaster of Paris for three or four weeks, the splint continuing the passive stretching. The correction can often be assisted by allowing the patient to walk about in the splint or plaster, and after a month of this treatment, when all rigidity is gone and stretching complete, massage and exercises may be begun. Very severe degrees of contracture, as in pes cavus and extreme valgus, will require something more than subcutaneous division of tendons and fascia, and it is far better to incise the foot extensively and divide all the resisting structures very precisely, than have recourse to tenotomy followed by severe wrenching with instruments which blindly damage quite unoffending structures. Open operation is infinitely preferable to subcutaneous tenotomy for lengthening such important tendons as the tibialis anticus and posticus, as by this means the tendon action is preserved and there is no fear of non-union or adherent union with scar tissue; also the tendons are available later if required for transplantation. Particularly in cases where this risk of weakness due to over-lengthening is great, it is always preferable to cut down on the tendon, lengthen it to exactly the desired amount and re-suture it. In the most severe cases, instead of a complete division of ligaments, some surgeons prefer to remove portions of the tarsus, in wedge-shaped sections or

whole bones, with the object of restoring the normal shape. Thus, for example, a wedge-shaped portion is removed from the region of the transverse mediotarsal joint to cure equino-varus; a wedge from the dorsum of the foot to correct cavus; and total excision of the astragalus for calcaneus deformity, flail foot, and severe equinus. Resection of bone leads to impairment or destruction of joint action, and is, therefore, in my opinion not such good practice, as gauged by the ultimate functional result, as the free division of all resistant ligaments and other structures. These major operations permit a partial correction at the operation, which is completed, if necessary, ten or fourteen days after, when the stitches are removed and the foot finally fixed up for a month or two in plaster of Paris. Having corrected the deformity, the means at our disposal for the prevention of its recurrence are along one or other of four main lines. We can maintain the position by appliances only; or if there is sufficient power left in the foot and leg we can redistribute that power and restore the muscular balance by some form of tendon transplantation. When the amount of power remaining is inconsiderable, or such that it cannot be usefully redistributed, either we can use the tendons of the paralysed muscles to act as fixation ligaments, fastening them to the tibia so that they keep the foot in dorsiflexion or hold up the heel, as the case may be; or, if we dislike tendon fixation, there remains the method of arthrodesis, or permanently ankylosing by operation the

joints which are uncontrolled and therefore a source of weakness. This operation is usually done on the ankle, the sub-astragaloid and the transverse medio-tarsal joints at one and the same time.

As operations for the correction of a deformity and the prevention of recurrence are carried out at the same time, it will be of advantage to describe in detail the operative measures for certain typical deformities. Let us first take a condition of severe dropped foot, or equinus, with dropping also of the forefoot producing a secondary cavus. The first stage in the operation is to correct completely the cavus or increase of arch, and restore the front of the foot to its proper alignment with the back part, before the equinus at the ankle-joint is corrected. A simple degree of cavus may be corrected by subcutaneous division or open resection of the plantar fascia, followed by stretching, and then the tendo Achillis can be lengthened subcutaneously at the same time, or possibly a week or two later. If the cavus is very severe, however, the best method is by a long incision beginning three or four inches above the ankle-joint and running down parallel to and just in front of the heel tendon; it then turns forward beneath the malleolus and passes along the inner border of the foot almost as far as the base of the big toe. This incision gives access to all the ligaments under the inner border of the foot—the calcaneo-scaphoid, scapho-cuneiform, and cuneiform-metatarsal, which should be freely and com-

pletely divided, and the central portion of the plantar fascia is resected. A second incision is made on the outer border of the foot, from the base of the fifth metatarsal to the back of the heel, parallel to the sole and at the level of the lower margin of the os calcis, to obtain access to the fascia, muscles, and plantar ligaments attached to the under surface of the os calcis, which are then completely detached and peeled forward after the method of Steindler. There is no need to make a complete horseshoe incision to detach the heel structures, especially in view of the inner incision already described. The division of the ligaments on the inner side of the foot and the turning forward of all the heel structures through the outer incision will allow any cavus, however severe, to be corrected. After such complete division of structures, a little manual stretching will easily overcome any tissue that may have been left undivided. Having corrected the cavus, the equinus may conveniently be overcome at the same time, the upper end of the inner incision giving access to the tendo Achillis. The heel tendon is longitudinally divided for some inches, lengthened by overlapping to the amount that is sufficient, and then resutured. For deep sutures, both here and in transplantations, I prefer to use fishing gut. The incisions are closed with fishing gut, and the foot put up in the best possible position, supported only by a simple back splint with foot piece. A fortnight later the stitches are removed, the precise correction determined, and

the limb fixed in plaster of Paris for another six or eight weeks. The patient may be allowed to walk on the plaster if a wooden sole be applied. In a foot showing severe cavus only, and associated with clawed toes but with no contracture of the heel tendon, the above operation is carried out as regards the arch, and at a subsequent stage the toes themselves are dealt with. For toes hyper-extended at the metatarso-phalangeal joints, in association with pes cavus, it is the custom to do a subcutaneous division of all the tendons of the extensor longus digitorum and extensor proprius hallucis, and subsequently to fix the toes down for a time in some light night splint and walking sock supplied with tapes to keep each individual toe in flexion. Sometimes the deformity of the big toe is treated by transferring the extensor proprius hallucis tendon to the first metatarsal just behind the head of the bone, the tendon being passed through a hole in the bone and sutured to itself. Tenotomy of the extensor tendons for clawed or hyper-extended toes only partially or temporarily relieves the deformity, as the tendons soon join up again, and in a few months the patient is very likely to be as bad as before. Even though it correct the hyper-extension of the toe at the metatarso-phalangeal joint, it does not correct the flexion at the inter-phalangeal joints, and therefore the two distal phalanges remain more or less permanently vertical and devoid of any function. The transference of the extensor proprius hallucis to the first metatarsal relieves the dorsi-

flexion of the metatarso-phalangeal joint, but causes the terminal phalanx of the big toe to drop into a flexed or vertical position, a condition not always appreciated by the patient who may thereby experience difficulty in putting on his boot. This operation is done with the idea of correcting or preventing cavus deformity, by making the extensor tendon lift up the distal end of the first metatarsal bone instead of dragging the big toe back into hyper-extension. By far the best operation for clawed toes is the transference of the long flexor tendon to the base of the proximal phalanx, leaving that of the flexor brevis digitorum to act on the middle phalanx. This operation corrects the flexion at the inter-phalangeal joints and permits the toe to lie flat on its palmar surface, while the transference of the long flexor power from the distal to the proximal phalanx allows flexion at the metatarso-phalangeal joint with the whole toe in a straight position, a function so valuable in walking, and comparable to the action of the lumbrical muscles in the fingers. A horse-shoe-shaped incision at the end of the toe and parallel to the nail border allows the long flexor tendon to be detached from its insertion, and through a separate very short incision in the mid-line just in front of the metatarso-phalangeal joint the long flexor tendon can be pulled out of the flexor tunnel. A small semi-circular incision on the dorsum of the toe just over the base of the proximal phalanx will permit a hole to be drilled through the base of the phalanx, through which the

long flexor tendon is passed after being threaded with fishing gut. The toe is then straightened in its inter-phalangeal joints, flexed at the metatarso-phalangeal, and, while held in a position of moderate flexion, the long flexor is pulled rather tightly through the hole in the basal phalanx and stitched to the split dorsal expansion of the extensor tendon on the top of the phalanx. This operation therefore necessitates three separate small incisions for each toe, but is quite simple and particularly efficacious. If there be much contracture of the extensor tendons, they may be lengthened by tenotomy at the same time, but this is not usually necessary as the flexor tendons can be pulled and stitched tightly enough to correct this extensor contracture. Instead of merely lengthening the extensor tendons by tenotomy, the operation of transference of the long extensor tendon to the corresponding metatarsal bone, which has been described in the case of the big toe, is done on all the other toes as well, or to simplify matters the four long extensor tendons may be inserted into two metatarsals. This operation is tedious; it may be of some assistance in preventing the recurrence of pes cavus but is not nearly so efficacious in correcting the deformity and promoting the valuable flexion function of the toes as the operation for transplantation of the long flexor tendons described above.

Turning now to some of the more important operations for tendon transplantation, we will first describe that used for paralysis of the gastro-

cnemius and soleus, an operation which I have done for some years, and of which I show you an example here. Through a small incision one or two inches in length in the middle of the outer aspect of the leg, the peronei muscles are examined to ascertain if they are functionable enough to make their transplantation worth while. In case both are good the peroneus longus is selected, or whichever appears the healthier. Through another small incision near the base of the fifth metatarsal bone the tendon of the peroneus longus or brevis, as the case may be, is divided and pulled up from below the outer malleolus into the incision in the leg. Next the foot is turned over, and a long vertical incision made from six inches above the ankle joint vertically downwards to the bottom of the heel, and midway between the tibia and the tendo Achillis. A transverse incision along the inner border of the foot joins the vertical incision at right angles, and extends forward almost to the base of the big toe. The tendo Achillis is split longitudinally, and then divided transversely so that it can subsequently be shortened by overlapping. For the moment the severed Achillis is turned aside. The upper and lower surfaces of the body of the os calcis are bared, and a vertical hole is drilled in the bone immediately in front of the insertion of the tendo Achillis, and large enough to take the transplanted tendons. The flexor longus hallucis and the flexor longus digitorum tendons are divided well forwards in the sole of the foot and pulled out of their tunnels

behind and beneath the inner ankle. The previously detached peroneal tendon is also pulled into the inner vertical wound, and all three tendons are threaded through the hole in the os calcis and pulled upon as tightly as possible. Before suturing the tendons in their new position, the two ends of the cut tendo Achillis are held in stout forceps, and then strongly pulled upon, overlapped, and sutured, to shorten the whole tendon and draw up the heel as far as it will possibly go. The tendo Achillis is sutured with fishing gut, and its shortening makes it act for a time as a ligament, to prevent strain falling upon the transplanted tendons before they are permanently adherent. There need be no limit to the amount of temporary equinus obtained by shortening the tendo Achillis. Similarly, the transplanted tendons are put under as much tension as possible, and then turned round and sewn to themselves or to the side of the os calcis. After suturing the skin, the foot is merely bandaged in equinus position, without plaster or splints, and thus retained for about six weeks, after which the patient is allowed to take a little weight on the toes. Massage and exercises may begin a week or two previously, and are continued in the walking stage. The artificial over-correction, or equinus, produced by the operation, will be found to disappear quite naturally when weight-bearing is resumed. If, as sometimes occurs, a considerable valgus element is found to be associated with the calcaneus deformity, maintained by the contracture of the external lateral ligament of the ankle-joint,

the latter should be freely divided to permit the heel to be drawn inwards or adducted, and the foot afterwards fixed for three or four weeks in a position of equino-varus. The operation for inversion or varus deformity due to unbalanced action of the tibialis anticus muscle consequent upon weakness of the peronei muscles is easy to do and singularly efficacious. Through a small incision at the inner border of the foot over the internal cuneiform bone and base of the first metatarsal, the tibialis anticus tendon is detached from its insertion. Another incision three or four inches long over the tibialis anticus tendon above the ankle-joint allows the tendon to be pulled up from beneath the annular ligament. Next, a small semicircular incision convex downwards is made on the outer border of the foot, to include the region of the base of the fifth metatarsal and the insertion of the peroneus brevis, but so planned that the skin incision does not lie over the line of the deep tendon suture. A long forceps is thrust up from the external incision, and the end of the tibialis anticus tendon grasped and pulled down to the base of the fifth metatarsal. While the foot is held in a position of extreme valgus and dorsiflexion by an assistant, the tendon is passed through a hole in the base of the fifth metatarsal and brought round and sutured to itself, or, if not long enough to permit this, to the peroneus brevis insertion or to the periosteum. Should any cavus deformity exist it can be corrected by enlarging the inner incision and dividing the

plantar fascia and the internal ligaments of the foot in the way described above for pes cavus. It is noteworthy that the cavus element usually associated with calcaneus deformity does not as a rule require any special correction, as, after the operation described above for calcaneus, the cavus gradually disappears when the patient takes weight again, showing that cavus associated with dropped heel must be largely due to the action of the long flexors of the toes. We describe next the operation employed in cases where the tibialis anticus, or the anticus and posticus together, is permanently paralysed, a defect which leads to the production of valgus or eversion deformity. The usual procedure is to utilize the peroneus longus for the tibialis anticus, dividing its tendon at the outer border of the foot, and then drawing it out of its tunnel up into an incision a few inches above the external malleolus, afterwards transferring it beneath the annular ligament to the region of the navicular, where it is passed through a hole in the tuberosity of that bone. The tendon is pulled strongly through the navicular, and, with the foot dorsiflexed and inverted as strongly as possible, is sutured to itself or to the periosteum. In paralysis of the tibialis anticus there is always a contracture of the tendo Achillis, and therefore care must be taken to lengthen the latter tendon before the peroneal suture is completed. Giving the peroneus longus tendon an insertion on the inner border of the foot does, it is true, adds dorsiflexion power to the foot, but the line of pull is so

oblique that, although the peroneal tendon dorsiflexes, it continues to abduct the foot, whereas the tibialis anticus muscle itself adducts as well as dorsiflexes. This operation, therefore, does not improve the adduction power, although it assists dorsiflexion. A better operation to substitute a defective tibialis anticus, by reason of giving adduction as well as dorsiflexion power, is that which utilizes the extensor proprius hallucis and the extensor longus digitorum to replace the tibialis anticus, while the peroneus longus is transferred to assume the toe extension function. A single long curved incision beginning five or six inches above the ankle joint, and running down rather to the outer side of the middle line of the leg and foot almost to the base of the metatarsals, will afford complete access. The tendons can be divided and an anastomosing rearrangement made above the anterior annular ligament if it be thought desirable to transplant healthy tendons into the paralysed tendon of the tibialis anticus. If, however, the preference is for a subperiosteal transplantation, the incision must be carried farther forwards on the dorsum of the foot, and a second incision will be required on the outer side of the foot to detach the peroneus longus tendon. Hitherto it has been a rule in transplantation operations for poliomyelitis not to insert healthy into paralysed tendons, but in view of the success that has attended the violation of this rule in analogous cases, such as in tendon transplantation for wrist drop after permanent

damage to the musculo-spiral nerve, it would seem that this contention can no longer be upheld. Although we have tried both methods, it is rather early yet to say from experience whether the insertion of healthy into paralysed tendons in infantile paralysis is quite safe to practise in transplantation operations in the foot. In cases where the tibialis posticus muscle also is paralysed, some substitute must be found for this muscle as well as the tibialis anticus. The incision should be along the inner border of the foot, and, after passing below the malleolus, ascend vertically up the leg for a few inches, parallel to the tendo Achillis and midway between it and the tibia. This gives access to the heel tendon, from which a longitudinal slip can be detached and grafted into the tibialis posticus tendon just above and behind the internal malleolus, while the remainder of the tendo Achillis can be split longitudinally and resutured after lengthening to the desired extent. Further power can be supplied in place of the tibialis posticus by dividing the tendon of the flexor longus digitorum in the sole, withdrawing it from its tunnel and carrying it forward again through that of the tibialis posticus to a new insertion into a hole in the tuberosity of the scaphoid bone. If both anticus and posticus are replaced at the same operation, this internal curved incision will give access to almost everything, but a further small incision on the outer side of the foot will be required to detach the tendon of the peroneus longus. All suturing should be made

with the deformity completely corrected, and the transplanted tendons as short as possible, the strain on the deep sutures being prevented by the fixation of the foot in over-correction in plaster of Paris. Tendon transplantation is applicable in preventing relapse of deformity only when sufficient residual power remains, and when the paralysed muscles constitute only a part of the group supplied by one or other of the two main nerves, the other components of that group being normal. As a general principle, therefore, we utilize healthy tendons to substitute only those of the same group, i.e., those supplied by the same nerve, whether the external or the internal popliteal. The only exception in the above operations is the utilization of the peroneus longus, supplied by the external popliteal nerve, to assume the rôle of a heel raiser (in the operation for calcaneus), a function controlled by the internal popliteal nerve. The peroneus longus is, however, a plantar flexor. Another important point to remember is that tendon transplantation is utilized only to prevent the recurrence of a deformity already corrected by other means, and not as a correcting influence itself. Either we must fully correct the deformity by some preliminary operation, or, if we correct deformity and transplant at the same time, the resistant tissues must be freely divided, to allow the foot to be put up in the over-corrected position in which it is desired that the transplanted tendon should act. When the muscular power remaining is insufficient to render transplantation practic-

able, as, for example, when the whole of the muscles supplied by one nerve, or the greater part of the group, is paralysed, we have at our disposal two methods other than transplantation whereby deformity is prevented. It does not appear to be of much use to transplant heel-raising and toe-flexing muscles to the front to act as dorsiflexors, and vice versa.

The first of the fixation methods referred to is that in which the paralysed tendons are cut away from their muscle bellies and sutured directly to the tibia two or three inches above the ankle joint, with the object of converting them into suspensory ligaments. In total paralysis of the muscles supplied by the external popliteal nerve, the tendons of the peroneus longus and tibialis anticus are inserted into a hole in the tibia, and under enough tension to hold the foot permanently in dorsi-flexion, preventing toe drop and inversion deformity, the foot being thus held at right angles with the leg. Similarly, a portion of the tendo Achillis may be fixed to the posterior surface of the tibia a couple of inches above the ankle-joint, to prevent heel drop in cases of total paralysis of the muscles supplied by the internal popliteal nerve. Personally I am not in favour of tendon fixation, and the manufacture of ligaments from paralysed tendons, as the tendons are very liable to stretch and permit early recurrence of the deformity. But some surgeons prefer this procedure to the methods of *arthrodesis*, or permanent fixation of uncontrolled joints by operations to

produce bony ankylosis. In order to stiffen joints permanently, the articular surfaces are deliberately denuded of cartilage, whereupon the joint is fixed for a couple of months in a good position in plaster of Paris, to render the ankylosis strong enough to permit weight bearing without risk of relapse of deformity. As a rule the operation is done only upon the ankle-joint, but I prefer in every case to include at the same time the sub-astragaloid and transverse mediotarsal joints. Access is by an external curved incision, similar to that employed in the operation of astragalectomy. When the external ligaments have been divided, the foot is forcibly adducted and the three joints completely opened up; full exposure cannot be obtained unless all the joints are quite dislocated. The whole of the articular cartilage is removed with a gouge and a resection knife, after which the foot is put up in the best possible position and remains so for a fortnight. Then the stitches are removed and the foot, now somewhat more consolidated, is fixed in plaster of Paris in the finally determined correct position. The operation gives a good stable foot, and the only trouble that is at all likely to occur afterwards is that from the still flail condition of the forefoot and toes, which may cause some difficulty in putting on the boot. This can be remedied either by having a boot laced well down to the toe-cap, or by shortening the extensor tendons of the toes. In place of ankylosing or arthrodesing operations in total paralysis of the foot, or instead of tendon transplantation in

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calcaneo-valgus deformity, some surgeons, following Whitman, prefer the operation of astragalectomy, contending that it produces a very serviceable foot. After the astragalus has been removed great care is taken to displace the foot well backwards in relation to the leg, and to retain it there until the new ankle-joint is consolidated. It is claimed that this operation gives a very stable foot in the conditions above mentioned, and, although allowing some movement, prevents relapse of deformity. In this country, however, the operation is not greatly in favour, the general impression being that tendon transplantation and arthrodesing operations are much more satisfactory.

INDEX.

Abdominal diathermy, for alimentary tract infection, 111
Adhesions, electro-therapy of, 51
Alcohol injection for trigeminal neuralgia, 79, 81
Alimentary tract, bacterial infection of, 102-108
  treatment of, 109-114
Amputation stumps, electrical treatment of, 48, 49
Anæsthesia, recovery stage of, 59, 61
  position and care of patient during, 60
Anæsthetics, after-effects of, 59
  depression and collapse following, 61
  excitement and struggling following, 62
  insanity following, 62
  pulmonary complications following, 66
  urine examination following, 65
  vomiting following, 62
Ankle, flail, cause and production of, 160, 161
  weak, cause of, 120, 121
    treatment of, 140
Ankle-joint, action and function of, 130, 154
  muscle groupings about, 154, 155
Arthritis, pyorrhœa alveolaris causing, 110
Arthrodesis, 195, 208
Astragaloid, action and function of, 132
Astragalus, action and function of, 120, 121
Asylums, congenital syphilitics in, 16, 17
  Wassermann reaction in, 16, 17
Ataxic symptoms of mental disorder, 14

B. coli, destruction of, by glycophilic streptococci, 108
  infection of intestinal tract from pyorrhœa alveolaris, 103
    treatment of, 109-114
Bile-ducts, congenital obliteration of, in newly-born, 36
  obstruction of, in familial jaundice, 34
Bile-staining in familial jaundice, 34
Bone injuries, electrical treatment of, 50
Boots and shoes, essential use of, 125, 145-147
  for deformities and disabilities of the feet, 126-129, 183, 184
  high and flat heels of, 125-129, 144
  ill-fitting, 145
    disabilities due to, 125-127, 136, 143, 144
  mechanical support provided by, 146, 147
Brain, syphilitic disease of, 16
  fits following, 8
Breast, cancer of, X-ray treatment of, 46, 47
Buhl's disease, 38
Bunions, 143

Cæcum, bacterial infection of, due to pyorrhœa alveolaris, 103, 110
Calcaneo-valgus deformity, cause and production of, 169
Calf-muscle paralysis, 159, 173, 174
Cancer, combined electrical and surgical treatment of, 46, 47, 53
Carbohydrate, effect on streptococcal infection of alimentary tract, 113
Cavus deformity, *see* Feet
Cerebral paralyses, causes of, 150
  deformities and disabilities of feet due to, 149, 150, 173
Charcot-Marie-Tooth's progressive muscular atrophy, deformities of feet due to, 152, 173
Children, boot and shoes for mechanical disabilities of feet of, 130
  flat feet of, 131
  "weak" feet and ankles of, 122, 140
Chill, causing trigeminal neuralgia, 76
Chloroform anæsthesia, vomiting following, 64
Chloroform poisoning, "delayed," 64
Cholangitis, in the newly-born, 37
Collapse, following anæsthesia, 61
Constipation, treatment of, 111
Corns, cause of, 143

Deafness, following syphilis, 7
Deformities, prevention of, 56

Deformities, *see also* Feet
Delusions, and real life experiences, connection between, 83
arising from jealousy, 93, 94
cases illustrating growth of, 83-94
dreams causing, 83, 84
facts developing into, 89
morbid, 83, 85
of syphilitics, 6, 7
sensory defects causing, 85
Dementia præcox, 94
Depression, following anæsthesia, 61
Diathermy, abdominal, for gastro-intestinal infection, 111
Diplegia, foot deformities associated with, 174
Dream conditions, and mental disorders, frequent relationship between, 83
obsessions in relation to, 88
Dreams, as cause of mental disorders, 83
Drugs, in exophthalmic goitre, 29
in trigeminal neuralgia, 79
Duodenum, bacterial infection of, from pyorrhœa alveolaris, 102, 103, 106, 115
treatment of, 109-114

Ear disease, syphilis causing, 7
Electrical tests, for paralytic deformities of feet, 186
Electrotherapy, combined with surgery, and observations on, 43-46
of bacterial infection of gastro-intestinal tract, 111
of cancer, 46, 53
of bone and joint injuries, 50, 51
of infantile paralysis, 55
of septic wounds, 41-46, 48, 49, 50
of tubercular conditions, 42, 44
surgical measures in combination with, 42, 43, 46, 50
see also X-rays
Epilepsy, syphilis in relation to, 7, 8
Equinus deformity, *see* Feet.
Erotic dreams, morbid mental conditions caused by, 83, 84
Ether anæsthesia, vomiting following, 64
Ethyl chloride anæsthesia, vomiting following, 64
Exophthalmic goitre, 19
drugs in, 29
fatal cases of, causes of death in, 23
results of treatment of series of cases of, 23-28
mortality rate from, 20, 21
prognosis of, 19-25
symptoms of, 22, 23
treatment of, duration of, 28

Fæces, streptococcal infection of, due to pyorrhœa alveolaris, 103, 112, 114
Familial jaundice, *see* Jaundice
Fears, morbid, 89, 90
see also Delusions
Feeblemindedness following syphilis, 8
Feet, action and function of, 116, 117, 130, 136, 176, 177
in men and women compared, 127
in relation to their strength, 124
adhesions of, treatment of, 133
anatomical structure of, 118-122
antagonistic muscle groups of, 154
attitude of rest or activity of, 117
breakdown of, predisposing cause of, 136
calcaneo-valgus deformity of, 169
cavus deformity of, 156, 159, 169, 170
nature of disability due to, 182
recurrence after correction, 164
treatment of, 194, 196, 197
contractures of, 156, 157, 169
deformities of, defect in growth of leg causing, 161
restoration of movements in, 122
disabilities due to static or mechanical causes, 116-148
disabilities of, treatment of, 137
dorsiflexion of, 154, 155
dropped, *see* equinus deformity, *following*
efficiency of, how estimated, 130
equino-valgus deformity of, cause and production of, 168
equino-varus deformity of, 158, 168, 172-4
nature of disability due to, 180
treatment of, 195
equinus deformity of, cause and production of, 157, 160, 173
nature of disability due to, 180
treatment of, 142, 195-197
exercises for, 137
flail, cause and production of, 160, 166, 170
mechanism of disability due to, 178
treatment of, 195
ligaments of, liable to strain, 127
mechanical disabilities, treatment of, 132-148
movements of, relative to the leg, 154
muscular action of, 116-118, 130, 154, 160, 176
painful, 133, 138
paralytic deformities of, 149-151, 162

Feet, paralytic deformities of, common types of, in infantile paralysis, 166, 169
difficulties and defects of function due to, 175
due to upper and lower neuron lesions, difference between, 153
electrical testing of, 186
examination of, 184-187
forcible rectification of, 165
functional or hysterical causes of, 153
how produced, 151, 155, 162
mechanical appliances for, 190
over-correction of, 192, 193
primary, 158
pseudo type of, 185
recurrence of, after correction, 163, 164, 193
secondary, 158
spina bifida causing, 172
splinting for, 162-3, 188-9, 190-2, 194
treatment of, 162-165, 188-210
types due to cerebral paralyses, 173
types due to infantile paralysis, 166-171
types due to neuritis, 171
plano-valgus deformity, 174, 175
plantar flexion of, 124, 125, 154
planus deformity of, cause and production of, 120, 121, 170
treatment of, 134
progressive deformity of, 138
pseudo-paralytic condition of, 161
splayed, 143
strain of, how lessened, 128
toe deformities, 142-145, 170-1, 198-9
trophic ulcers of, 179
valgus deformity of, cause and production of, 120-21, 159, 169, 204
nature of disability due to, 181
treatment of, 134, 140, 194
varus deformity of, 170
functional, 153
operative treatment of, 203
"weak," cause of, 120, 121, 131
mechanism of, 177, 178
treatment of, 122, 137, 138
weight-bearing and leverage action of, 117, 155
see also Flat foot; Heel, etc.
Fits, in relation to syphilis, 7, 8
Flail-foot, 160, 166, 170
Flail-joints, mechanism of disabilities due to, 177, 178
Flat foot, cause of, 120, 131, 145
treatment of, 122, 131, 134, 139
Fractures, electro-therapy of, 50, 51
Friedreich's disease, foot deformity associated with, 152, 173

Gasserectomy, for paroxysmal trigeminal neuralgia, 79, 80
results of, 81
Gasserian ganglion, alcohol injection into, 79, 81
Gastrocnemius paralysis, nature of disability due to, 159
operative treatment of, 200, 201
Gastro-intestinal tract, bacterial infection of, from pyorrhœa alveolaris, 102, 115
treatment of, 109-114
General paralysis, cause of, 9, 12, 13
characteristics of the patients, 10, 11
diagnosis of, mistakes in, 9
direct connection between syphilis and, 13
fits of, 10
juvenile or adolescent, 15
latent causes of, 13, 14
locomotor ataxy in relation to, 14
morbid heredity of, 16
neurotic inheritance in, 13
prognosis in, 10
progress of the patient, 15
racial characteristics of, 12
syphilis in relation to, 9, 12, 13
without signs of mental disorder, 11
Genu valgum, cause of, 120
Glycophilic streptococci, 107
Gums, streptococcal infection of, 102, 108

Hallux rigidus, 143
Hallux valgus, 142, 144
Hammer toe, 142
Heel, deformities of, 169
function of, 125
dropped, 159
cause and production of, 168
nature of disability due to, 180, 181
short tendons of, test for, 130, 131
treatment of, 132
Heels, of boots and shoes, function of, 126-129, 144
Hemiplegia, foot deformities associated with, 174
Heredity, syphilis and, 16
trigeminal neuralgia in relation to, 75
Hypochondriasis, morbid mental growths causing, 86
Hysteria, development into mania, 95

Idiocy, congenital syphilis in relation to, 15, 16
Infant, *see* New-born infant
Infantile paralysis, combined surgical and electrical treatment of, 55
deformities and disabilities of feet due to, 151, 166-169
treatment of deformities due to, 188, 191
Influenza, symptoms of general paralysis following, 13
Insane jealousy, suspicion developing into, 93
Insanity, adolescent, 95
ataxic symptoms of, 14
congenital syphilis causing, 15
following anæsthesia, 63
general paralysis and, 10
locomotor ataxy and, 14
morbid mental growths in relation to, 83, 85, 97
syphilis and, 1, 2, 15
Intestinal antiseptics, 112
infection, treatment of, 109, 114
Intestines, bacterial infection of, from pyorrhœa alveolaris, 102-115
treatment of, 109-114

Jaundice, congenital splenomegalic hæmolytic, 38
infective, in new-born infant, 37
during pregnancy, 31, 32, 33
simple, physiological or idiopathic, of newly-born, 36
Jaundice, familial, of newly-born, 31
ætiology of, 33
diagnosis of, 35, 36
maternal toxæmia theory of, 32, 37,38
morbid anatomy of, 34
prognosis of, 35
syphilis in relation to, 34, 38
treatment of, 38
Jealousy, insane, suspicion developing into, 93
Jejunum, bacterial infection of, from pyorrhœa alveolaris, 103,
treatment of, 109-114
Joint injuries, electricity in treatment of, 50

Keloid, electrical treatment of, 49, 53
Keratitis, and gasserectomy, 79, 80, 82
Knock-knee, cause of, 120

Leg, antagonistic muscle groups of, 154
defect in growth of, causing foot deformity, 161
foot movements in relation to, 154
Leg, muscles of, action and functions of, 154
shortened, disability due to, 183
Liver, enlargement of, in new-born infant, 36, 37
Locomotor ataxy, general paralysis in relation to, 14
insane interpretations of morbid sensations due to, 14
syphilis in relation to, 14
Lung complications, following anæsthetics, 66

Malignant disease, combined electrical and surgical treatment of, 46, 47
Mania, febrile stage of syphilis followed by, 6
hysteria developing into, 95
Mediotarsal joints, action and function of, 120, 121, 135
Melæna neonatorum, 38
Mental conditions, morbid, dreams causing, 84
Mental decay, following syphilis, 5
Mental disorders, and dream conditions, frequent relationship between, 83
ataxic symptoms of, 14
cases of general paralysis without signs of, 11
congenital syphilis causing, 15
exophthalmic goitre in relation to, 23
fits in relation to, 7
neurasthenia developing into, 95
syphilis as a psychical cause of, 2-5
Mental growths, morbid, 83
destructive or malignant variety of, cases illustrating, 94
invading variety of, 88
simple variety, 85
Mentally defectives, congenital syphilis in relation to, 16, 17
Muscle groups, antagonistic, 154
Muscles, electrical treatment of, 57
Mysophobia, and syphilis, 5

Neck glands, tuberculous, combined surgical and electrical treatment of, 44, 45
Neuralgia, trigeminal, *see* Trigeminal neuralgia
Neurasthenia, developing into mental disorders, 95
general paralysis and, 6
syphilis in relation to, 6
Neuritis, deformities of feet due to, 152, 171
New-born infant, congenital obliteration of bile ducts in, 36

New-born infant, grave familial jaundice of, 31-39
infective jaundice of, 37
melæna of, 38
septicæmic infection of, 38
simple, physiological, or idiopathic jaundice of, 36
Nitrous oxide anæsthesia, vomiting following, 64

Obsessions, growth of, cases illustrating, 88-91
morbid mental growths in relation to, 88
Optic neuritis, syphilis and, 6, 7
Orthopædic surgery, object of, 165
Os calcis, action and function of, 121
Ovarian function, x-rays regulating, 53, 54

Paralysis, infantile, 55
Paralytic deformities of the feet, 149
Paranoias, characteristics and cases illustrating, 95-97
Persecution, simple facts giving rise to ideas of, 95, 96
Pes, *see* Feet
Peritoneal adhesions, prevention of, 51, 52
Petit-mal in old cases of syphilis, 8
Plano-valgus deformity, cause and production of, 174, 175
Plantar flexion, mechanism of, 125
Planus deformity, *see* Feet.
Plastic surgery, electricity in, 50
Poliomyelitis, anterior, *see* Infantile paralysis
Popliteal nerve paralysis, treatment of, 208
Pregnancy, jaundice during, 31, 32, 33
Pyorrhœa alveolaris, infective conditions due to, 104, 115
treatment of, 109-114
intestinal infection due to, treatment of, 109, 114
effects, 101-115
Psychic analysis, possibilities of, in treatment of delusions, 97
Ptosis following syphilis, 9
Pulmonary complications following anæsthesia, 66

Radio-surgery, risks of, 54
Radio-therapy and radio-surgery, difference between, 53
See also X-rays.

Scar tissue, and X-rays, 45
Scar tissue, electrical treatment of, 49
Sensory defects, delusions due to, 85
Septic wounds, combined surgical and electrical treatment of, 41, 43, 46, 48, 50
Sexual dreams, morbid mental conditions caused by, 84
Shock, following anæsthesia, 61
Shoes, *see* Boots and shoes
Skin affections, electro-therapy of, 42-43
tuberculous affections of, combined surgical and electrical treatment of, 42, 44
Soleus paralysis, nature of disability due to, 159
operative treatment of, 201
Spina bifida, deformities and disabilities of feet due to, 152, 172
Spinal cord, electro-therapy of, 55, 57
Spinal nerves, injuries to, deformities of feet due to, 150, 152
Spirochæte, general paralysis in relation to, 9
Spleen, enlargement of, in new-born infants, 36, 37
Splenomegalic hæmolytic jaundice, congenital, 38
Standing, effect on musculature of the feet, 117-119
Starches, in treatment of gastro-intestinal infection, 112, 113
Stomach, bacterial infection of, from pyorrhœa alveolaris, 102-115
treatment of, 109-114
effects of anæsthetics on contents of, 65, 66
Streptococcal infection, bio-chemical considerations, 105
dietetic treatment, 113
vaccine therapy of, 114
Streptococcal vaccine, for intestinal infection, 114
Streptococci, glycophilic, 107
destruction of *B. coli* by, 108
infection of intestinal tract by, from pyorrhœa alveolaris, 102-115
treatment of, 109, 114
Sub-astragaloid joint, action and function of, 132
Surgery, electricity and x-rays in combination with, 41, 43, 45-47
radio-, and radio-therapy, difference between, 53, 54
Syphilis, as a cause of familial jaundice, 34, 38
as a psychical cause of mental disorder, 2-5
congenital, as cause of insanity, 15
asylum experience of, 16, 17
epilepsy in cases of, 7, 8

Syphilis, congenital, familial jaundice in relation to, 38
direct connection between general paralysis and, 13
ear disease resulting from, 7
epilepsy and, 8
feeblemindedness following, 8
fits in relation to, 7, 8
general paralysis in relation to, 9, 12, 13
inheritance of, 16
insanity in relation to, 1, 6, 15
locomotor ataxy in relation to, 14
mental decay following, 5
moral effects of, 3, 5
morbid heredity of, 16
neurasthenia following or associated with, 6
optic neuritis following, 6, 7
parental, mental degeneracy in children and, 17
physical causes of, 7
sensory defects in relation to, 6
Syphilophobia, 3, 90
physical causes of, 7
psychical causes of, 3, 7

Taka-diastase, for gastro-intestinal tract infection, 112
Teeth, septic, bacterial invasion of gastro-intestinal tract due to, 102, 110, 115
Tendo Achillis, contracture of, 132, 171, 172
deformity due to, 167
treatment of, 204
transplantation of, 201, 202
Tendon fixation in deformities of the feet, 195
transplantation, 200-207
Tenotomy, for deformities of feet, 194
Tibialis anticus paralysis, 158
deformity due to, 166, 167
operative treatment of, 204
Toes, clawed, causes and production of, 170, 171
nature and disability due to, 183
operative treatment of, 198, 199
deformities of, 142-145
disabilities due to, 179
treatment of, 198, 199
dropped, cause and production of, 174
Toes, hammer-, disability due to, 183
hyper-extension of, 145
trophic conditions of, 179
Trigeminal neuralgia, age incidence, 73
bilateral, 80
causes of, 70, 71
character of pain of, 72, 77
chill causing, 76
chronic paroxysmal, 68
heredity as a factor in, 75
left and right side incidence, 71
pathology of, 69, 70
sex incidence, 71
treatment of, 79-82
warnings of onset of, 76
Tuberculin, 44
Tuberculosis, treatment of, 43-45
Tuberculous affections, combined surgical and electrical treatment of, 42, 44
X-ray treatment of, 45, 46

Vaccine therapy of bacterial infection of gastro-intestinal tract, 114
Valgus deformity, *see* Feet
Varus deformity, *see* Feet
Vomiting, post-anæsthetic, causes, prevention and treatment, 62-66

Walking, action of feet during, 117, 136
improper habits of, disabilities of feet due to, 136
mechanism of the action of, 177, 178
passive attitude in, 117-119
Wassermann test, 15, 16, 17
Winckel's disease, 38
Wounds, combined surgical and electrical treatment of, 41, 43, 48, 50
x-ray and electrical treatment of, 43-46

X-rays, action of, on tissue, 53
dosage and frequency of, 47
in malignant disease, 46, 47
in surgery, indications for, 45-46, 47
in tuberculous affections, 45, 46
post-operative treatment by, 46, 47
regulating ovarian function, 53, 54
surgical measures in combination with, 41, 43, 46
See also Electrotherapy

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